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FOCAL EPILEPSY AND THE STUDY OF CORTICAL FUNCTION.[1]

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THE subject of this lecture is particularly appropriate because the material was made available largely by the foresight of Sir Hugh Cairns[2] and Sir Charles Symonds. They induced the Service medical departments to set up during the last war a neurological and neurosurgical organization designed especially to deal with brain wounds. After the war the Medical Research Council supported a Head Injury Bureau sited in Oxford and organized largely by Dr. Ritchie Russell, which could maintain contact with these patients and follow their medical and social welfare. The bureau also had access to a specialized in-patient rehabilitation unit, and the whole constituted a unique and valuable organization for long-term assessment and follow-up.

It is from this source that Dr. Ritchie Russell and I have been able to study over the past ten years traumatic epilepsy arising in small penetrating brain wounds. Apart from the intrinsic interest of post-traumatic epilepsy, it is also a potential source of information about cerebral and particularly cortical localization; for if the site of origin of the epileptic discharge is known, then a study of the content of the fit and particularly the initial phase or aura can give us some indication of the function of that site. Moreover, the march of events in the fit can give additional information about the elective pathways leading from the area of initial firing. This approach has been very profitably exploited by electrical stimulation of the cortex and more recently of subcortical structures, particularly by Wilder Penfield (1954) and his school, and by Earl Walker (1959), and their work will be well known to you. When one makes use of traumatic focal epilepsy in this way, there are two assumptions on which any deductions we make about cerebral function are based, and it is important to recognize them as assumptions, however valid we may consider them. Firstly, the site of origin of the discharge is taken to be in close proximity to the site of injury. The evidence for this is broadly that the removal of the injured and nearby areas will often abolish the attacks, stimulation may induce the naturally occurring pattern of the attack, and in addition the content of the attack is often consistent with the general picture built up over many years and from a variety of clinical and experimental sources of

[1] The third Hugh Cairns Memorial Lecture, delivered in Adelaide on March 3, 1960.

[2] Owing to restriction of space, some personal references to Sir Hugh Cairns in the lecture as delivered had to be omitted.

the function of the site injured. This evidence is not entirely unequivocal, but it justifies the assumption. I have not mentioned the recording of localized electrical abnormality, particularly focal spiking, from the supposed site of discharge. The opportunity for such recording and even for routine electroencephalography occurred only rarely in our cases, and I shall not therefore be referring to it. Its value as a further lead to the site of origin of epileptic discharge is considerable and undoubted; but the view that the occurrence of a spike focus indicates precisely the origin of the fit is now no longer entirely acceptable. High-voltage spikes can be recorded at times when no overt clinical epileptic activity of any sort is occurring. Removal of cortex in which spiking is occurring does not always abolish the clinical fits; and occasionally as one area of spiking is removed, so another previously silent may appear. The search for a spike focus as the main guide to cortical excision is not now so keenly pursued, though it remains a useful part of the evidence. Fortunately, in the majority of clinical cases such activity is in fact recorded in close proximity to some macroscopic abnormality, which lends some further support to our first assumption.

Secondly, we assume that the effects of local epileptic discharge bear a close relation to those of the physiological discharge occurring in the area at times when it is functionally active in normal behaviour; and in addition we take it that the pathways pursued by the epileptic discharge reflect those of the physiological. Direct evidence for these—recording regional electrical cortical activity and tracing the pathways it takes during varying types of normal behaviour—is as yet lacking. Indirect evidence is provided by the constancy of results of stimulating a given area by a variety of means, and within limits by the results of excision. So far as the path of discharge is concerned, the clinical march of an attack and the course of electrical activity following direct stimulation of the site both demonstrate elective pathways presumably to some extent anatomical and available for use in normal cerebral function. It is likely, of course, that the pathway for any epileptic discharge is to some extent artificial and unphysiological, and that this may also be reflected in the content of the attack. For the present, however, it is justified to use these assumptions, though as we learn more of cerebral physiology we may have to return to question them.

One further point in considering focal epileptic discharge and focal electrical stimulation as evidences of cerebral function: the latter has obvious advantages in localizing the site of origin of the abnormality, but it can be argued that the epileptic discharge arising from the natural activity of nerve cells—albeit damaged ones—and without direct outside intervention is more likely to reflect aspects of a normal function of the area.

Some General Problems of Ætiology.

In addition to the special information provided by the study of focal epileptic wounds, there are some general questions about epilepsy which such a study encounters. I should like briefly to discuss three of these. Firstly, why does epilepsy occur? Secondly, why does it occur when it does? Thirdly, given the common factor of trauma, does it occur more readily in one site than another?

The occurrence of traumatic epilepsy used to be unhesitatingly linked with the extent of cerebral damage, and particularly the more chronic and widespread damage provided by sepsis and subsequent scarring. This view is now no longer so tenable. During the last war, early and improved techniques of surgical *débridement* and the enormous reduction of sepsis with antibiotics must have considerably reduced secondary brain damage. Yet the incidence of fits in 820 cases of brain wounds which we have studied is running at about 43% at a five-year follow-up (Russell and Whitty, 1952). This can be compared with Ascroft's (1941) figure of 45% for the 1914-1918 War and with Baum's (1930) of 44% from German sources. Both these were culled 10 to 15 years after wounding, and our own figure is likely to be slightly higher by that time. The same story is told by patients with known sepsis in our series. Here, even in those cases of prolonged brain hernia, fungus or abscess, epilepsy occurs in only 52%—a not significant rise on the over-all incidence.

The actual extent of the wound does appear to have some influence, since cases of persistent hemiplegia, representing more widespread cerebral damage, have an incidence of 66%. This figure must be viewed critically, however, because hemiplegia indicates not only more extensive damage, but often damage to the central parts of the cortex, where, as we shall see in a moment, the regional incidence of epilepsy is higher in any case.

The constitutional factor must also play some part. Evidence from family histories in this group of 820 cases is not available; but in a study of early traumatic epilepsy occurring within one month of wounding (Whitty, 1947) the familial incidence was clearly significant, and Cobb's (1932) review of epilepsy in closed head injuries also emphasizes this. The tendency for epilepsy to approach 50% in a variety of brain lesions—trauma, tumour and abscess—is perhaps explained by the interplay of a general, constitutional, factor of cerebral excitability with local injury. If the distribution of the general factor in the population at risk follows the normal bell-shaped curve, then epilepsy following cerebral trauma may tend to occur only in the 50% of the population which form the right half of the curve. Such a view makes constitution a paramount factor in ætiology.

The time factor in traumatic epilepsy presents two problems—the general time of the onset of the condition, and the precise timing of any one attack. So far as the latter is concerned, we should probably regard all epilepsy as to some extent reflex epilepsy. The physiological state at the site of origin of the attacks, and particularly the pattern of afferent stimuli reaching it, together with the synaptic set of its efferent pathways, will determine whether firing occurs. Of these factors we already recognize and make clinical use of certain general ones, such as lack of sleep, lowered blood-sugar level and emotional upsets, as conditions likely to precipitate an attack and presumably affecting the *milieu interne* of the epileptic focus. More rarely, our patients may themselves notice some specific precipitating stimulus which they thereafter avoid. As knowledge of cerebral physiology increases, we shall doubtless recognize more often such specific afferent impulses, both exteroceptive and enteroceptive (which may include emotional ones), as precipitators of attacks, and as we do so our therapy will become more rational.

The time of onset of the condition after wounding can be seen in Table I. In a study of 356 cases, about 70% of attacks started in the first twelve months. These figures agree substantially with those of two other series from the 1914-1918 war, shown in Table II. In nearly three-quarters of cases epilepsy begins within the first year, and after three years the likelihood of developing epilepsy is slight, although in ours and in others' experience the occasional case may occur up to fifteen or twenty years later. Such very late cases may well have a quite incidental factor of vascular pathology in their origin;

TABLE I.

Interval between Wounding and First Fit in 356 Cases of Post-Traumatic Epilepsy in Brain Wounds.[1]

Interval.	Number of Cases.
<1 month	47
1–6 months	94
6–12 months	62
1–2 years	49
>2 years	28
Not known	76

[1] See acknowledgements.

but the grouping of the mass of cases in the first two years calls for explanation. Progressive scarring does not seem an adequate explanation, both for the reasons given above and because if the time interval for onset in various regions of wounds is measured it is found to vary. In Figure I, occipital, temporal and central wounds have their greatest incidence at six months, with a small secondary rise about two years. Frontal wounds, on the other hand, show a slower rise to a maximum incidence at about one to two years. The same picture is shown in epilepsy following cerebral abscess. It would seem

TABLE II.

Interval between Wounding and First Fit in Three Series of Cases of Post-Traumatic Epilepsy following Brain Wounds.[1]

Interval.	This Series.	Ascroft.	Baumm.
0– 1 month ..	47	19	
1– 6 months ..	94	22	122
6–12 months ..	62	15	
1– 2 years ..	49	10	42
2– 5 years ..	28	9	39
Total ..	280	75	203

[1] See acknowledgements.

likely to be based on the anatomy and function of the region injured. It may be relevant, for instance, that the frontal regions differ from the others in being a main

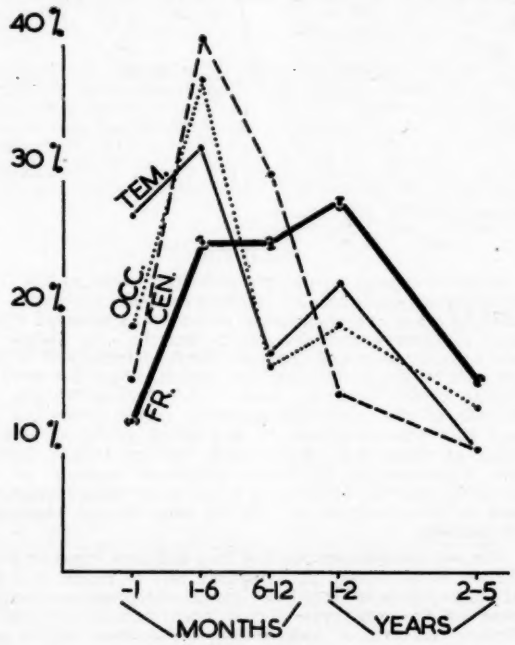

FIGURE I.

Cen. = wounds in motor and pre-motor areas and parietal lobes; Fr. = wounds in frontal areas anterior to area 6; Tem. = wounds in temporal lobe; Occ. = wounds in occipital lobe. See acknowledgements.

pathway for the diffuse thalamo-cortical projections, as contrasted with the more detailed thalamic projections to the central areas.

Apart from this time factor, the over-all incidence differs in different regions. In Table III, 601 cases in which evidence for localization of the wound was satisfactory are analysed. Incidence is highest in the Rolandic area, and becomes less as the frontal and occipital poles are approached. Another feature which our figures show is that wounds on the medial aspects of the hemispheres are significantly less likely to produce fits than those on the lateral aspects. The incidence is 35% in the former and 56% in the latter. There seems clear evidence that there are regions of relatively high epileptic potentiality, and again this is likely to depend on the functions and connexions of the areas concerned.

TABLE III.

Site of Wound and Incidence of Fits.

Site of Wound.	Totals.	Subjects with Fits.
Prefrontal and inferior frontal	160	63 (39%)
Motor and premotor	127	70 (55%)
Parietal lobe	170	111 (65%)
Temporal lobe	71	27 (38%)
Occipital lobe	73	28 (38%)
Total	601	299 (48%)

Distribution of Wounds Causing Epilepsy.

When the distribution of brain wounds is under consideration, it is convenient to chart them in some manner so that they can be compared. This has been done in the majority of our cases from the pre-operative lateral X-ray plates, using a device (Russell, 1947) which measures the centre of bone defect in relation to two standard coordinates, the nasion-inion line and a perpendicular to this passing through the centre of the bone defect. Only small superficial wounds are suitable for this type of charting, and even in these the impression may be misleading in individual cases. A number of such distribution charts will be shown; but I would emphasize that their value is only as an indication of general concentration of wounds in a given area, and they may be misleading in considering the individual case. In the latter, detailed information is required about direction and depth of foreign body and bone fragments, and of any early or persistent neurological deficit; fortunately this is usually available in our cases. For purposes of clarity, wounds within 5 cm. of the sagittal line are charted as a halo to the main skull outline. Figure II shows the site of epileptic (marked with a cross) and non-epileptic (marked with a circle) wounds in some 600 cases. It shows that there seem to be no obvious areas which are entirely free from epilepsy, and brings out the relatively low incidence in the medial areas represented in the halo. The diagonal line represents the position of the central sulcus.

One of the chief interests of the distribution of these wounds lies in its correlation with the content and especially the aura of the attack. By aura I mean simply that part of the attack which is consciously experienced by the patient. In this sense, focal attacks and auras are largely interchangeable terms, and are used as such throughout this discussion.

Sensory and Motor Fits.

In Figures III and IV, 33 patients with a variety of sensory fits and 51 with motor fits are charted. The legends to the figures indicate the nature of the attacks. One striking feature of these is that although the sensory attacks concentrate in the post-Rolandic region, the motor attacks, especially the clonic ones, are also well represented in this region. It is usually considered that the clonic element of a fit is essentially a cortical phenomenon, and when motor cortex is largely destroyed a tonic fit results. The distribution in these cases, with adversive and tonic fits occurring most commonly when motor cortex is directly damaged, lends some support to this view. It seems equally likely, however, that a largely deafferented motor area will more readily discharge clonically, since

the physiological studies of Brazier (1951) and others have shown an enhanced tendency to spontaneous discharge in isolated neurons or groups of neurons. In the majority of these cases of post-Rolandic wounds there was in fact some permanent sensory defect implying a degree of deafferentation. The view that deafferentation is important is also supported by the methods occasionally employed to terminate or abort a clonic fit. Some patients discover for themselves that the application of a strong sensory stimulus to the part first affected—forcible tension to the muscles affected, attempted strong voluntary contraction and less often cold, heat or pain—may end the attack. By contrast, attempts to abort sensory attacks by these methods are in our experience unsuccessful.

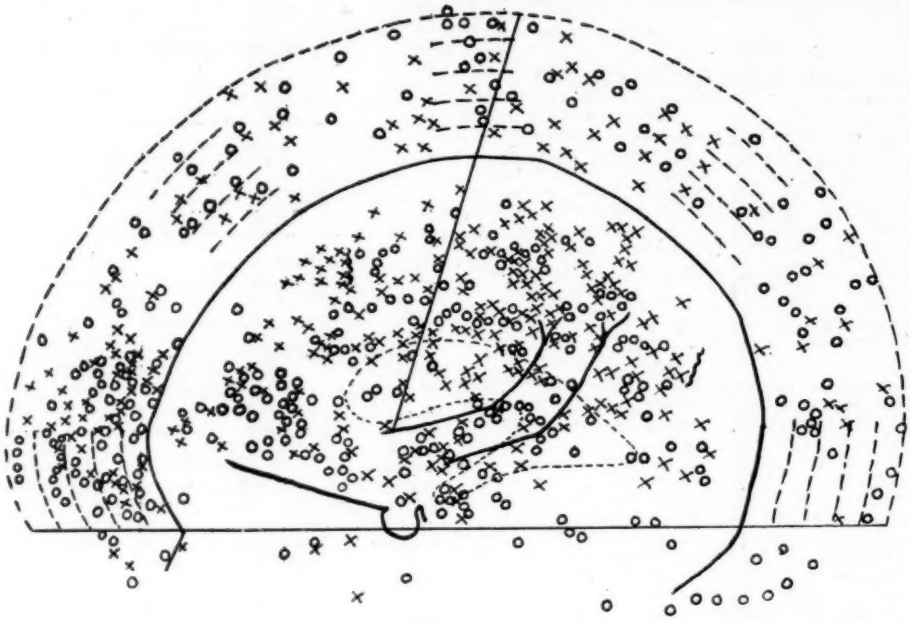

FIGURE II.
Distribution of epileptic (X) and non-epileptic (O) wounds.

Of the sensory attacks, one interesting feature was their clear localization; both their site of onset and their march were usually clearly and accurately described. This suggests that the area concerned with appreciation of the body image remains functioning normally, despite the abnormal discharge from an area which forms part of its usual source of information. Another point of special interest was the occurrence of painful auras. These were described in four cases and again were well localized, though not so precisely as non-painful sensations. Thermal auras also were well localized. Cold was experienced, but never described as unpleasant. Heat, on the other hand, varied from a pleasant warmth to a severe causalgia. Cortical representation of pain has often been denied as a result of the work of Henry Head. However, these cases seem to afford evidence in favour of it.

Dyphasic Fits.

Thirty patients had an unequivocal dysphasic element to their aura, and the distribution of their wounds is shown in Figure V. Some had some degree of permanent dysphasia between attacks, and this then became more dense; in many, however, there was no detectable interictal speech defect. Of the whole group, more seem to be represented behind the central sulcus than in front, and the distribution is rather above and anterior to the conventional siting of Wernicke's area. The appearance of dysphasia as the onset of an aura rather more commonly with anterior wounds probably only represents the fact that a motor loss of speech is more readily appreciated by the patient and onlookers than a sensory one. It proved difficult in many cases to decide from their description and even from those of witnesses whether a sensory dysphasia occurred, and it is possible that some patients with ill-defined auras of being muddled or confused were really describing sensory dysphasia.

Visual Fits.

Wounds causing visual fits constitute some of the most interesting examples of forced cortical activity. In addition, they are phenomena which many patients find it easy to observe and describe accurately. In Figure VI, 44 of these cases are charted. The wounds tend to concentrate in the occipito-parietal region, where the so-called higher visual cortex is sited. There are strikingly few wounds of the immediate calcarine region producing fits, and this was confirmed in a detailed study of over 60 cases of visual fits (Russell and Whitty, 1955). Indeed, the incidence of fits with unilateral wounds of the occipital pole was lower than in any other region examined, and we were tempted to look for some special anatomical explanation.

We can distinguish positive and negative types of visual fit, the latter consisting of sudden loss of vision in a part of or the whole field, of brief partial obscurations, shadows, etc.; but the two types do not appear to have any characteristic pattern of localization. Moreover, both could occur together in one fit, the usual sequence being an initial negative phenomenon passing on to a positive phenomenon. We found no example of positive phenomenon passing to negative, though, of course, when positive hallucinations occurred in a part or the whole of a field this obscured normal vision. The content of positive fits varied widely. Many were simple unformed hallucinations. A few were complex hallucinations of remembered scenes which could be clearly described. These might be static, like a still film, or there might be movement and action going on. In general these were in natural colours, although a few were described as black and white. It

is of special interest that these could occur in one half-field, sometimes with preservation of vision in the other half. In addition to those patients who could describe the hallucinations, there were some who were certain that they had had a fully formed visual hallucination and that it was a familiar scene, but were unable afterwards to remember what it was. They thus showed a classical *déjà vu* phenomenon. Wounds in these cases of complex hallucination appeared to be both temporal and occipito-parietal. This fact suggests that, as far as visual imagery is concerned, some recall mechanism linked with memory may be situated in the parietal region in addition to the well-recognized temporal lobe siting of

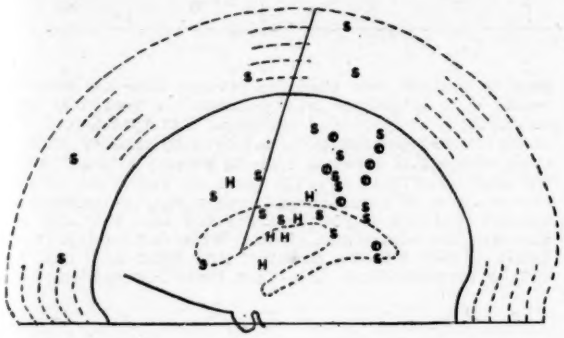

FIGURE III.

Distribution of wounds causing sensory fits. S = positive sensory; H = thermal; ⊅ = inhibitory or negative sensory. See acknowledgements.

the lesion in *déjà vu*. Two cases (mentioned in a previous study of visual fits—Russell and Whitty, 1955) are worth describing in some detail, as they illustrate the way in which cortical function may be deployed in the visual sphere.

CASE I.—This patient had had a right-sided brain wound with a metallic foreign body entering through the orbit

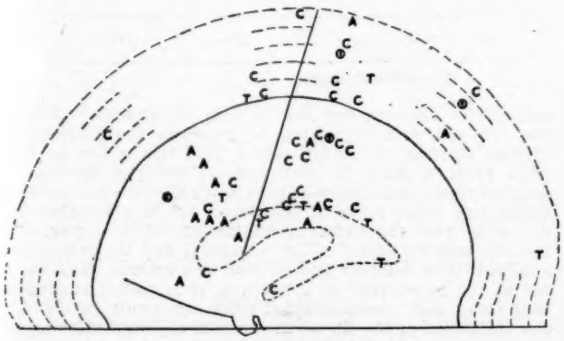

FIGURE IV.

Distribution of wounds causing motor fits. A = adversive; C = clonic; ⊅ = inhibitory; T = tonic. See acknowledgements.

and destroying the globe. It had ended up in the right posterior temporal cortex. He had a persistent left upper quadrantanopia, but no other neurological signs. Early after wounding he had *grand mal* attacks without any aura. Later an aura occurred of a sudden black spot in the centre of the left half-field, which spread rapidly to involve the whole left field. At times, instead of the loss of vision, the left field would be filled with coloured balls of light, which changed as he looked at them to tiny replicas of men, which after some repetition of the attacks he recognized as mirror images of himself. More rarely, the initial black spot aura would occur, but immediaely be filled with a life-sized head and shoulders picture of himself. This could be made to follow movements he himself made. All these auras had at times led on to *grand mal* attacks.

Here a simple hallucination became secondarily organized into a complex autoscopic one. The phenomenon is bizarre, and it is worth mentioning that the patient's evidence seemed entirely reliable. Moreover, such a build-up of autoscopic hallucinations from more simple ones has been previously recorded (Hecaen and Ajuriaguerra, 1952). It suggests that an area of primary discharge in the visual cortex must fire into that concerned with the body image. In this, as in all recorded

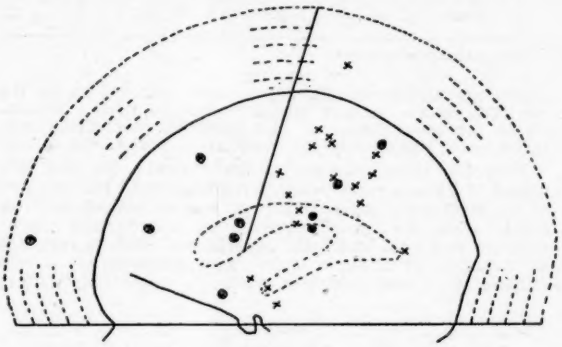

FIGURE V.

Distribution of wounds causing aphasic fits. X = fit containing aphasic element in aura; ⊗ = fit initiated by aphasia.

cases of autoscopy, the visual hallucination of the self is a mirror image, again suggesting that it is the visual exteroceptive element of the body-image mechanism which can be involved in a relatively isolated fashion.

CASE II.—This left-handed patient, aged 28 years, had had a right occipito-parietal brain wound with early left hemiparesis, sensory loss and hemianopia. Later there

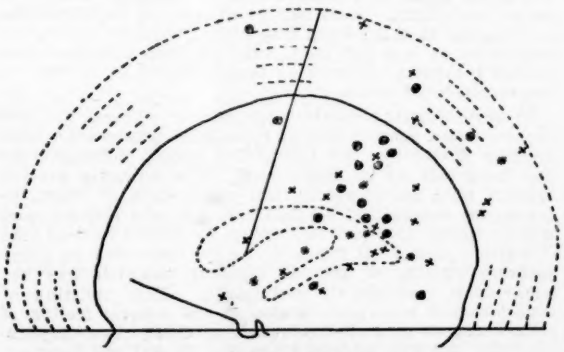

FIGURE VI.

Distribution of wounds causing visual fits. X = positive visual aura; ⊗ = negative visual aura; • = aura of remembered scene.

was some attention defect only in the left half-field, with minimal sensory loss in the left arm and leg. After this he had attacks of clonic movement of the face leading to loss of consciousness. In some attacks facial movement was followed by aphasia and then loss of consciousness. He also had attacks in which his thought suddenly seemed to get out of control, and he felt as if in a dream he was back in Khartoum during the war. He saw and recognized friends around him and heard them talking. The experience was accompanied by strong emotion of just the sort he had felt when isolated in Khartoum in 1940, at a time when he thought he should be in England

TABLE IV.
Site of Onset of Focal Motor and Sensory Fits.

Site.	Motor Clonic.	Motor Tonic.	Adversive Tonic.	Motor Inhibitory.	Sensory Positive.	Sensory Inhibitory.	Totals.
Face, head and eyes ..	15	4	12	—	6	3	40
Upper limb	26	8	—	5	21	9	69
Trunk	3	1	—	—	1	4	9
Lower limb	9	1	—	2	4	1	17
Totals	52	14	12	7	32	17	135

[1] See acknowledgements.

where his family was taking a more active part in the war. During the attacks, which lasted less than a minute, vision for objects around him was obscured. These episodes were occasionally the aura to a *grand mal* attack.

Here the visual element of the patient's fits was only a part of a much more complex reminiscence. He appeared to be back once more living in a remembered environment, with the accompaniment of appropriate visual, auditory and even emotional experiences. This is certainly an example of firing a memory mechanism in a very holistic way, and contrasts with the more fragmentary activation which still retains obvious qualities of memory shown by limited visual hallucinations. It is interesting to note that in this second case during some of his attacks consciousness was not fully lost (his wife described him as dazed); but to himself he seemed really to be back in the remembered scene.

TABLE V.
Detailed Site of Origin of Motor and Sensory Fits in 126 Cases.[1]

Site.	Number of Cases.	Site.	Number of Cases.
Face	20	Thumb	2
Head and eyes ..	20	Fingers	8
Leg	11	Hand	31
Foot	6	Arm	28

[1] See acknowledgements.

In reviewing the considerable variety of focal fits and auras—motor, sensory and of the special senses—one most striking feature is the ability for normal consciousness and behaviour to be fragmented. The epileptic process appears able, as does electrical stimulation at times, to throw one segment of cortical function into activity, and within limits this activity gives to the individual the experience of normal function; at the same time in many instances much of normal cerebral function and its behavioural correlate is maintained. This splitting of consciousness with preservation of the general feeling of self-awareness and individuality certainly has implications for the psychologist interested in cerebral function. It is noteworthy that the complexity and range of sensory phenomena are far greater than those of motor phenomena. In general the latter are simple, unorganized and a caricature of the great flexibility and dexterity of normal motor life. Moreover, we have never encountered organized speech as an epileptic phenomenon though compulsive thoughts and ideas usually stereotyped for the individual have been noted as part of an aura.

Pathways of Epileptic Discharge.

Having considered the general site of discharge in differing auras, we may consider briefly its subsequent path as illustrated by the content and march of events in the fit (Russell and Whitty, 1953). We have already noted, for instance, that negative visual phenomena may pass to positive, but that the reverse does not seem to occur; that aphasia is more commonly a secondary than an initial phenomenon of the aura; and that arrest of a motor fit may sometimes be achieved by sensory stimulation, though this does not apply to sensory attacks. When we consider (Table IV) 135 cases in which the clinical site of onset of auras could be accurately determined, it appears that first the upper limbs and then the head and face are the commonest sites. When we look in more detail (Table V), we find that the head and face are equally represented; in the upper limb, however, the hand and digits account for nearly 60% of attacks, while in the lower limb the position is reversed, the majority of attacks starting in the leg rather than in the foot or toes. This must depend to some extent on the amount of cortical representation and its relative distribution between digits and other parts of the limb; but it must also link up with the functional organization of the part—the amount and flexibility of its movement and the consequent availability of afferent and efferent connexions. Indeed, so far as the upper limb is concerned, it is rather surprising that index and thumb are not more commonly involved in the initiation of the fit, as the results of electrical stimulation in man and animals would lead us to expect.

TABLE VI.
Spread of Sensory and Motor Auras.[1]

Spread.	Sensory Auras.	Motor Auras.
To sensory	Same 22 Other 3	6
To motor	28	Same 27 Other 42
Fit ceased	20	55
To loss of consciousness	4	41
To other	3	3
Totals	80	174

[1] See acknowledgements.

If we look at the clinical march of the attack (in Table VI), where 254 examples of sensory and motor auras are given, there are several interesting differences between sensory and motor attacks. Sensory fits tend to spread readily to motor (28 out of 80); when they remain sensory, they may spread in extent, but they rarely change their original sensory modality. Twenty-two of 80 remained the same, only three changed; about a quarter of them ceased spontaneously while still confined to their primary manifestation; only four of 80 proceeded to loss of consciousness. On the motor side, however, only six of 174 spread from motor to sensory; 69 remained motor, but of these no less than 42 changed their motor characteristics (clonic, tonic, adversive and inhibitory) as the attack progressed. Of the motor sequence, while clonic to

tonic was most common, clonic and probably tonic to inhibitory was recorded, but the reverse was not seen; motor fits ceased spontaneously, as did sensory fits, in rather more than one-quarter of instances, but no fewer than 41 of 174 passed on to loss of consciousness, in striking contrast to the sensory march. This may reflect the anatomical arrangement of the cortex in which motor attacks start, because we shall see, when considering fits with loss of consciousness without aura, that they, too, had an anatomical grouping. These findings certainly suggest that there are elective pathways for impulses from one area of cortex to another. The most striking is the ready passage of sensory to motor. This is what

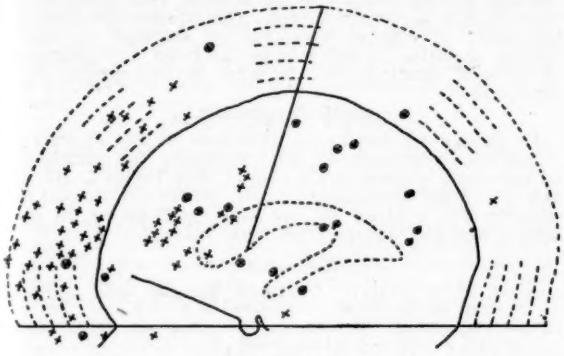

FIGURE VII.

X = fits always without aura; ⊗ = persistent neurological signs.

common sense would suggest, since motor performance must be continually guided, controlled and largely instigated by sensory impulses. The paucity of spread from one type of senory modality to another must also reflect basic

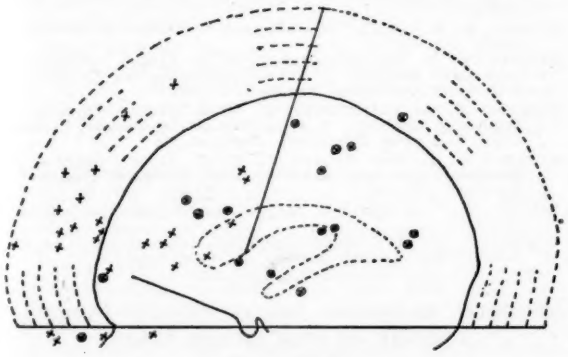

FIGURE VIII.

X = *grand mal* always without aura; ⊗ = persistent neurological signs.

cortical organization. It would suggest that the specific cortical areas stimulated must play some part in the quality of sensation experienced, even if (as some workers on the physiology of sensation would argue) the pattern of sensory impulses arising at the periphery is also important; and it would imply a relatively rigid afferent segregation of areas subserving different modalities. For instance, the tendency for inhibitory phenomena to follow positive motor attacks, while they more commonly precede positive visual attacks, is also noteworthy. In normal vision some inhibitory process must be important if newer visual images are not to be obscured by persistence of what has gone before. In the motor sphere, however, inhibition will be an end result of movement, and further action will be initiated via sensory stimuli.

Fits Without Aura and Status Epilepticus.

We have considered fits with various auras; but just as interesting are those cases in which there is no aura, and the patient passes directly to loss of consciousness, usually with some generalized motor accompaniment either excitatory or inhibitory. In Figure VII, 75 such cases are charted. All these patients had fits always without aura. There is an undoubted concentration in the frontal and pre-frontal regions, and in those cases in which the site of the wound was post-central, it is worth noting that the majority of patients had persistent motor and sensory signs, usually of hemiplegic distribution. It is therefore possible that in them the normal spread of discharge

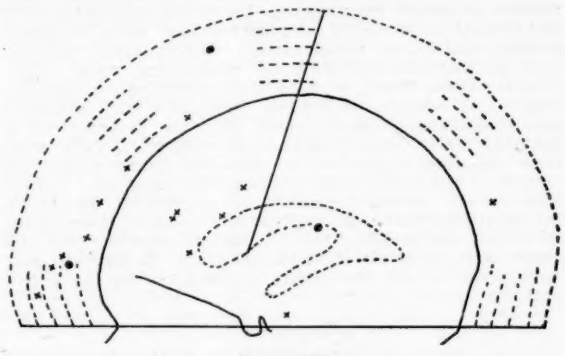

FIGURE IX.

X = akinetic attacks; ⊗ = persistent neurological signs.

which would evoke an aura was prevented by destruction of axons. If we break down the attacks into excitatory and inhibitory or akinetic (Figures VIII and IX), the same pattern of distribution is seen. Clonic fits in this group without aura are so few as to be unsuitable for

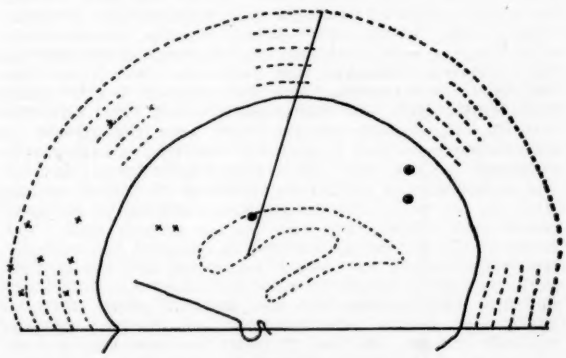

FIGURE X.

X = status epilepticus always without aura; * = status epilepticus with aura; ⊗ = status with persistent signs.

group charting; but they, too, are all frontal. Amongst those patients who never had an aura are 11 who had status epilepticus. There are an additional three whose status was preceded by an aura, and these 14 are charted in Figure X. Here again in all but two cases the wounds are in the frontal region. This tendency for status to occur especially with frontal lesions has been noted also in a mixed group of cases associated with tumour, abscess and trauma drawn from a two-year intake of a combined neurological and neurosurgical department previously reported (Whitty, 1950). All the 29 patients with status epilepticus had entirely or predominantly frontal lesions. A frequent occurrence of status in association with abscesses of the frontal region has also been noted.

The relatively high incidence of status in frontal lesions may only reflect the fact that *grand mal* epilepsy without aura is in Hughlings Jackson's sense the "local sign" of frontal cortex. Generalized status can thus be viewed as epilepsy partialis continuans of the frontal areas, and as comparable with sensory or focal motor epilepsy partialis continuans fired from the post-Rolandic or pre-Rolandic areas; it is not necessary to postulate some special perseverative or repetitive quality in discharge from the frontal region. However, since most focal auras do not lead as a routine on to *grand mal*, it does imply that discharge from the frontal areas has a more ready access to those subcortical centres which mediate generalized convulsions. It also appears that these frontal areas have elective pathways for discharge to regions concerned with the normal maintenance of consciousness, since the most striking feature of frontal epilepsy is the abrupt onset of loss of consciousness with no intervening aura. The frontal areas have a rich cortico-cortical projection system; in addition they have connexions with thalamic and hypothalamic nuclei, and there is evidence of a probably direct fronto-mesencephalic pathway. They must thus constitute a part of the two-way diffuse projection system of the brain stem reticular formation and cortex. This would certainly provide a mechanism by which consciousness could be rapidly and readily interrupted. It might also provide the anatomical substrate for the rapid and complete involvement—both inhibitory and facilitatory—of all the bodily musculature in the *grand mal* fit.

Conclusion.

If we review the sidelights cast on cortical function by the study of focal epilepsy, the general picture that emerges is of the cortex as the organ of discriminative consciousness. This can be fragmentarily activated by the epileptic process, so that the function of one part may be displayed inappropriately and out of context. Such isolated activation is not apparently incompatible with continued and seemingly normal activity of other cortical areas. This activity can on the sensory side be extremely complex and well coordinated within its own limits; particularly striking examples of this are seen in the focal visual fits which constitute complex remembered experiences. Such artificial and fragmented functioning can embrace thoughts and emotions which to the individual experiencing them may appear largely indistinguishable from his normal mental activity. Epileptic activity can thus irrupt into and alter the stream of consciousness without necessarily rendering it subjectively abnormal. This must have important implications for our understanding of the organization of normal mental life. On the motor side, however, the activity is far more coarse and rudimentary than on the sensory side. The motor component of psychomotor or temporal lobe epilepsy, which approaches most nearly to normal motor behaviour, is in a special category, since it represents not direct epileptic involvement, but the indirect effect of interruption of normal consciousness and memory by the epileptic process. In general, motor fits form only a crude caricature of the finesse of well-executed skilled voluntary motor behaviour. This is to be expected if we consider that sensory impulses must be the guide to fine motor activity, for such activity when originating in motor pathways themselves is then likely to be relatively coarse. Moreover, even when focal motor fits occur with an initial sensory aura, initiated so to speak by sensory impulses, they are still formed of crude and ill-organized movements; this emphasizes that sensory guidance is a continuing part of normal motor behaviour. The cortex appears increasingly as an afferent organ, and we are reminded of Pavlov's conception of it as a central analyser.

In addition to this differential discriminative consciousness, there is also a more crude all-or-none awareness, without which the discriminative functions of the cortex do not operate. This also can be involved in epileptic discharge, and the cortical representation of this mechanism appears to be in the frontal areas. Unlike the discriminative functions, however, epileptic firing here appears only to induce negative response—loss of awareness. It would seem probable also that in this instance the cortex operates via some subcortical mechanism.

Interesting as such observations are in their own right, their main importance must, I think, lie in the help they can afford us in filling in the complex picture of normal cerebral activity. There is, however, an important *caveat* to any simple correlation of epileptically induced and normal cerebral functioning. When epileptic discharge occurs, it must have some effect on the function of other areas to which the discharge passes, even if no overt change can be readily detected. At least the nuances of cerebral function may thus be altered, even when seemingly normal activation occurs. The evidence provided by the study of epilepsy can thus be accepted as useful interim material on our way to the fuller understanding of cerebral function, but it must be interpreted with discretion.

Acknowledgements.

My thanks are due to the publishers of *The Journal of Neurology, Neurosurgery and Psychiatry* for permission to reproduce Figures I, III and IV and Tables I, III, IV and VI. I am also grateful to Dr. W. Ritchie Russell for allowing me to draw on our joint publications on this subject and for supplying me with Table V.

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ARTERIAL GRAFTING FOR OCCLUSIVE ARTERIAL DISEASE OF THE LOWER LIMBS.[1]

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OCCLUSIVE ARTERIAL DISEASE of the lower limbs is an extremely common affliction. However, in spite of extensive research, prevention is not possible, and until recent years, treatment has been unsatisfactory. Vasodilator drugs are useless in cases of structural arterial disease; sympathectomy, although extremely valuable in the relief of symptoms mainly due to ischæmia of skin, does not cure the intermittent claudication from muscle

[1] This paper was presented in brief at the Second Asian-Pacific Congress of Cardiology, Melbourne.

ischæmia, and is often of no avail in saving a limb with imminent or established gangrene.

When it was shown that blocked arteries could be replaced by vessels transplanted from another subject, great hopes were raised that this method would prove of value in the treatment of occlusive arterial disease. Although early reports on this method were favourable, its limitations were soon apparent. A relatively small percentage of patients with occlusive arterial disease were suitable for operation, and many grafts ceased to function within a period of months or a few years.

It is opportune at this time, some 10 years after its inauguration, to assess the place of this treatment in occlusive arterial disease. In this paper we present the results of the 62 arterial grafting operations performed on 52 patients during the past six years with a variety of techniques, and in the light of this experience, assess the therapeutic value of this operation and its indications.

Short Historical Survey.

Pioneer work in the grafting of blood vessels in animals was performed by Alexis Carrel in 1902. However, with the exception of the work of Lexer (1912), who used a vein graft for bridging a defect in an artery, the technique of vascular grafting was not used in man for about 50 years. Meanwhile, Murray (1939) found that the success rate of vascular anastomoses in animals could be greatly increased by the use of the recently discovered anticoagulant drug, heparin.

The first successful homografts in the human were reported by Gross and his associates (1948), who used grafts placed in a solution containing serum and salts and stored just above freezing point — a method which gave better results, as judged by tests of viability using tissue cultures, than others investigated (Peirce *et alii*, 1949). Other methods of preserving grafts developed successfully by other workers included rapid freezing and storage at low temperatures (Hufnagel and Eastcott, 1952) and freeze-drying (Marrangoni and Cecchini, 1951). Meanwhile, Fontaine and Hubinont (1950) reported successful results in treating arterial occlusions by the use of vein grafts.

Arterial grafting was used successfully for occlusive arterial disease of the peripheral vessels such as the femoral and politeal arteries (Eastcott, 1953; Brown *et alii*, 1953) and also in traumatic lesions of the arteries resulting from warfare (Jahnke and Seeley, 1953).

The procurement and preparation of the arterial grafts by an aseptic technique presented a major difficulty. It was later found possible to use grafts sterilized chemically with beta-propiolactone (Szilagyi *et alii*, 1954) and with ethylene oxide (Hufnagel *et alii*, 1953). Since viability of the tissue was not necessary for grafting, synthetic prostheses were tried. As a result of the demonstration that vinyon "N" cloth could be successfully used as an arterial prosthesis in dogs (Vorhees *et alii*, 1952; Blakemore and Vorhees, 1954), this substance was successfully used in the repair of the abdominal aorta in man (Blakemore and Vorhees, 1954). Other workers followed with the use of various synthetic materials — nylon, orlon and dacron (Deterling and Bhonslay, 1955, 1956; Edwards and Tapp, 1955, 1956; Knox and West, 1957). One of the most recent substances used for this purpose is teflon (Girvin *et alii*, 1956; Harrison, 1957; Merendino *et alii*, 1958), which has proved superior to other synthetic materials (Harrison, 1959; Edwards and Lyons, 1959).

In the earlier operations, the occluded section of artery was excised and the graft inserted by end-to-end suture to the severed ends above and below. It was later appreciated that excision of the diseased artery was not necessary and also possibly harmful by destroying collateral vessels, and that a more satisfactory result could be obtained by a side-to-side anastomosis (Crawford *et alii*, 1955; Linton and Menendez, 1955). At present our standard grafting operation for occlusive arterial disease is the use of a teflon prosthesis inserted by a side-to-side (shunt) technique.

Material and Methods.

Selection of Patients.

Our criteria for selection of patients can be considered under three headings: (i) the severity of the symptoms; (ii) the general condition of the patient; (iii) the anatomical site and extent of the arterial block. In general, patients considered most suitable for operation were those with severe symptoms not amenable to other treatment (for example, with claudication at a walking distance of less than 200 yards, or with ischæmic rest pain persisting in spite of sympathectomy), whose general condition was good. It was not advised for patients with mild symptoms, or with symptoms which could be relieved by the simpler operation of sympathectomy, or for patients in poor general condition due to old age, ischæmic heart disease or cerebral vascular disease. In such patients operation would present undue risk, and the benefits of an improved circulation of the leg might add little to general enjoyment of life. Usually, only patients satisfying these two criteria (severe symptoms, good general condition) were investigated further to discover whether the arterial lesion was amenable to surgery. In some instances, when the limb appeared in jeopardy and arterial grafting appeared to offer the only means of saving it, the criteria concerning the general condition of the patient were relaxed somewhat, and operation was performed in spite of advanced age and the presence of other disease. The operation has been performed on only about 8% of patients with occlusive arterial disease of the legs referred to us in the past six years.

Preoperative Assessment.

In addition to full clinical examination, further tests of the patient's general condition included electrocardiography and estimation of the fasting blood sugar level, the blood urea content and the serum cholesterol content.

Routine clinical examination of the limbs included observation for trophic changes, temperature changes and postural changes and oscillometry. Arteriography was performed by the use of a percutaneous technique. The contrast medium was usually injected into the common femoral artery on the affected side, but in some instances in which the pulsation in this vessel was poor or absent, it was injected through a catheter introduced along a special flexible guide into the common femoral artery of the opposite side according to the technique described by Seldinger (1953).

Subjects.

Operation has been performed on 52 patients, of whom 45 have been subjected to a single operation in one limb, three to operations on both legs and four to multiple (two to four) operations on the one limb. Other data concerning these 52 patients and the indications for operation are shown in Table I.

Males outnumbered females in the ratio of 6:1, as would be expected from the greater incidence of arterial disease in males. With one exception, the patients were aged over 40 years, with the peak incidence in the 60 to 69 year decade; very old patients were excluded. The commonest disease of the arteries was atherosclerosis obliterans. Eighteen patients had hypertension, but this was not usually severe and was of the atherosclerotic type. Only three patients gave a history of symptoms of coronary artery disease; in general, a history of a recent coronary artery occlusion or the presence of angina pectoris was considered a contraindication to operation. Five patients were diabetic.

Owing to the risks of thrombosis, embolism and rupture, the presence of a popliteal aneurysm was regarded as an indication for operation regardless of the severity of ischæmic symptoms. In the other cases the ischæmic symptoms were severe and interfered materially with

the patient's way of life. The commonest symptom was intermittent claudication. Eight patients had features of imminent or actual gangrene, and arterial grafting seemed to offer the only hope of saving the limb. Arterial grafting was not commonly advised for distal ischæmic symptoms (coldness and rest pain), as these are usually relieved by sympathectomy.

Surgical Treatment.

It is not our purpose in this paper to describe details of surgical technique, and only general reference to methods will be made. In the earlier operations, the grafts comprised human arteries removed aseptically, rapidly frozen and stored at −5° C.; more recently, teflon

TABLE Ia.
Ages of Patients.

Years.	Number of Subjects.
30+	1
40+	9
50+	11
60+	22
70+	8
80+	1

arterial prostheses have been used. At first, the blocked segment of artery was excised and replaced by a graft inserted by an end-to-end suture technique. Later, the blocked segment was left *in situ* and the block circumvented by a graft inserted by a side-to-side technique. In the early phase, short grafts of 10 to 20 cm. (4 to 8 in.) were commonly used. More recently, longer grafts of about 45 cm. (18 in.) have been used to by-pass not only the blocked section but the whole of the atheromatous superficial femoral artery. Short grafts have been reserved for cases in which the pathological condition is localized, as in popliteal aneurysm. The types of grafts are depicted diagrammatically in Figure I, which also shows the number of each type and the number of patent grafts achieved. The sites of grafts inserted by the different methods are shown in Table II. In the early phase of the treatment, heparin was administered by an intravenous drip method at a rate of approximately 1000 units per hour for the three days after operation, suture of the wound being postponed until the end of this time. Anticoagulant therapy was continued by the administration of ethylidene dicoumarol in a dose sufficient to maintain the prothrombin level between 10% and 40% of normal. Later in the series intravenous heparin therapy was omitted, the only heparin given being 2500 units injected at operation into the arterial tree distal to the occluding clamps; apart from this, anticoagulant therapy was limited to administration of ethylidene dicoumarol, commenced on the day prior to operation.

Follow-up Investigation.

Anticoagulant therapy with ethylidene dicoumarol has been continued for a minimum of six months and in many cases indefinitely.

Assessment of the operative results was made by observations of the condition of the limb, examination of the pulses and oscillometry prior to the patient's discharge from hospital (usually a fortnight after operation), approximately four weeks after operation and thereafter at intervals of six months.

Results.

Operative Success Rate.

Reestablishment of circulation and discharge of the patient from hospital with a functioning graft have been achieved in 53 of 62 operations, as shown in Figure I.

In these successful cases, symptomatic relief has been dramatic. The patients have generally been completely

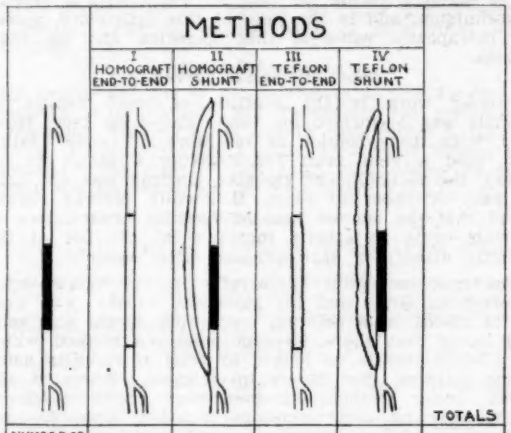

FIGURE I.

relieved of symptoms — both claudication from muscle ischæmia and rest pain, coldness and paræsthesia from skin ischæmia.

Information concerning the nine operative failures is presented in Table III. It is apparent that in seven of the nine cases there were unfavourable factors (second operation, double operation, poor distal vessel, radiation fibrosis). In one case there was a technical error, and in one no apparent cause for the failure.

Mortality and Morbidity.

There has been no immediate operative mortality. One patient died from chronic wound sepsis about three months after operation. In four of the cases in which a satisfactory graft was not established, the limb was amputated. In one of these, in which the post-operative course was complicated by severe wound infection and hæmorrhage from the graft, the amputation was pre-

TABLE Ib.
Diseases Present and Indications for Operation.

Arterial Disease.	Number of Patients.	Other Diseases.	Number of Patients.	Indications for Operation (62 Grafts).	Number of Patients.
Atherosclerosis obliterans	43	Hypertension[1]	18	Aneurysm (with or without ischæmic symptoms) ..	6
Popliteal aneurysm	5	Diabetes	5	Claudication	28
Traumatic thrombosis	2	Coronary artery disease ..	3	Claudication and distal ischæmic symptoms	16
Radiation fibrosis	1			Distal ischæmic symptoms ..	4
Dissecting aneurysm	1			Gangrene or incipient gangrene	8

[1] Blood pressure more than 160/100 mm. of mercury.

TABLE II.
Methods and Sites of Grafts.

Method.	Site.					Total.
	Aorto-Femoral and Aorto-Popliteal.	Ilio-Femoral.	Common Femoro-Popliteal.	Superficial Femoro-Popliteal.	Popliteal.	
Homograft end-to-end ..	—	1	—	10	5	16
Homograft shunt	1	—	18	11	—	30
Teflon end-to-end	—	—	—	—	2	2
Teflon shunt	1	1	12	—	—	14
Total	2	2	30	21	7	62

cipitated by operation; in the other three it was due to the arterial disease present before operation.

Post-operative morbidity has been low, and most patients have been discharged from hospital about two weeks after operation. In two cases, healing was delayed for several weeks. Four patients have suffered from hæmorrhage associated with arterial grafts, one patient having two episodes.

In one case already discussed as an operative failure, the graft was removed and the leg amputated before the patient's discharge from hospital.

In another (already mentioned as the case of delayed healing), in which hæmorrhage occurred in hospital, the graft was repaired and continued to function for 12 months and then thrombosed. A second grafting operation with a teflon prosthesis was then performed. This graft functioned for three months, when the patient suddenly developed a hæmatoma of his thigh of obscure origin, with obstruction of the graft; it was not possible to reestablish an adequate blood flow in the leg, and amputation became necessary.

In the other two cases the bleeding occurred a few weeks after the patients' discharge from hospital, and although the grafts were repaired, early thrombosis occurred.

Slight œdema has occurred occasionally in the limb subjected to operation. In one case it was severe and persisted for several weeks, necessitating the patient's readmission to hospital.

Survival of Grafts.

The survival times of the 62 grafts are shown graphically in Figure II, in which the grafts are grouped according to operative technique.

The over-all follow-up status at the present time is as follows:

Grafts blocked before discharge	9	
Grafts blocked since discharge	17	53
Grafts patent until time of patient's death	4	53
Graft patent until patient lost from supervision	1	53
Grafts still patent	31	53
Total	62	

Only half of the original 62 grafts are still patent. A better estimate of graft prognosis is obtained by comparing the number of grafts patent after a certain period with the possible number, as is done in Table IV, Part A. This, however, may not give an accurate present-day prognosis, as the outlook may be improved by new technique. Table IV, Part B, therefore, shows similar

TABLE III.
Information Concerning Operative Failures.

Graft Number.	Patient's Age. (Years.)	Sex.	Indication for Operation.	Arterial Disease.	Other Diseases.	Site.	Method.[1]	Comment.	Result for Patient and Limb.
11	65	M.	Claudication, distal ischæmia.	Atherosclerosis.	——	Superficial femoro-popliteal.	E.E. (H).	Second operation, wound infection, hæmorrhage.	Amputation.
16	74	M.	Incipient gangrene.	Atherosclerosis.	Hypertension	Superficial femoro-popliteal.	S. (H).	Technical error (arteriovenous anastomosis).	Amputation.
17	49	M.	Claudication.	Atherosclerosis.	—	Superficial femoro-popliteal.	S. (H).	Second operation.	*In statu quo.*
18	61	F.	Incipient gangrene.	Atherosclerosis.	——	Common femoro-popliteal.	E.E. (H).	Very poor distal vessel.	Amputation.
33	71	M.	Claudication, distal ischæmia.	Atherosclerosis.	——	Common femoro-popliteal.	S. (H).	——	*In statu quo.*
35	47	M.	Claudication.	Atherosclerosis.	——	Commom femoro-popliteal.	S. (H).	Endarterectomy below graft.	*In statu quo.*
37	63	M.	Claudication.	Atherosclerosis.	——	Common femoro-popliteal.	S. (H).	Two limbs operated on one session.	*In statu quo.*
39	50	F.	Claudication.	Atherosclerosis.	Diabetes, Hypertension	Common femoro-popliteal.	S. (H).	Very poor distal vessel.	Amputation.
49	66	F.	Distal ischæmia.	Radiation fibrosis.	Hypertension	Common ilio-femoral.	S. (T).	Massive scarring from radiation, infection.	Death of patient.

[1] E.E., end-to-end; S., shunt; H., homograft; T., teflon.

figures with the early end-to-end period (prior to December, 1954) excluded. There is a progressive decrease in survival rate with increased time from operation, the value being approximately 50% after three years. Unfortunately, it is not possible to include figures for the most modern technique with the use of a teflon prosthesis, as this is too recent.

Comparison of Methods.

The incidence of blocks for the main types of operation is shown in Table V. This shows that the highest incidence of immediate blocks occurred with the shunt-homograft technique. However, subsequent blocks were more frequent in the end-to-end grafts, so that the over-all position with regard to patency favours shunt

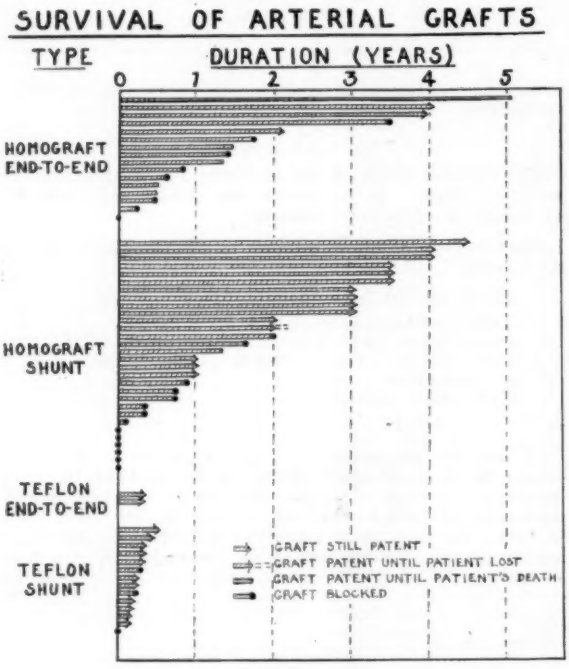

FIGURE II.

grafts. This is also illustrated in the tabulation showing the number of surviving grafts in relation to the possible. The figures are improved for all periods by excluding the early end-to-end period. The method using a teflon prosthesis compares favourably with the other methods as regards immediate graft patency, but it is too early to compare this method with those using homografts in respect to long-term graft survival.

Discussion.

After the early optimistic reports on arterial grafts for occlusive arterial disease, it became apparent that the late results were not as good as the immediate ones. Thus Hoye and Warren (1956) found that of 16 patients who left hospital with patent grafts, nine had closed grafts at follow-up examination (usually within the first six months). Szilagi *et alii* (1956) found that of 97 patent grafts, 21 became occluded in the subsequent 33 months; and Jahnke (1958) found that five of seven patients treated by arterial grafts for traumatic lesions had occluded graphs at follow-up examination some four years later. Szilagi *et alii* investigated the cause of late failure by post-operative angiography and by exploration and biopsy in cases of occlusion. They concluded that the most common factor in late graft failure was the extent and severity of the preexisting arterial disease; but in a proportion of cases there was evidence from the angiograms of degenerative changes in the arterial substitute. These papers are based on the older methods of grafting using homografts and an end-to-end suture technique.

De Bakey *et alii* (1958) have recently reported on their results in a large series of 448 cases of aorta-iliac

TABLE IV.
Graft Survival in Relation to Period following Operation.

Survival Period.	A: Complete Series.		B: End-to-End Period Excluded.	
	Number of Grafts.	Number Patent.	Number of Grafts.	Number Patent.
One-year potential (before December 31, 1958) ..	46	27	34	22
Two-year potential (before December 31, 1957) ..	38	17	26	15
Three-year potential (before December 31, 1956) ..	35	14	23	12
Four-year potential (before) December 31, 1955) ..	21	5	9	4
Five-year potential (before December 31, 1954) ..	11	1	—	—

occlusion, and in 353 cases of occlusive disease of the femoral and popliteal arteries. The second group of patients were mainly operated on by a by-pass technique using a synthetic prosthesis (nylon or dacron). The immediate success rate was 86% (almost identical

TABLE V.
Incidence of Blocks According to Operative Methods.

Method.[1]	Total Number of Grafts.	Blocks.		
		Immediate.	Subsequent.	Total.
E–E (H)	16	1	8	9
S (H)	30	7	7	14
E–E (T)	2	0	0	0
S (T)	14	1	2	3

[1] See footnote to Table III.

with our own results), and was approximately the same for the different methods of excision and replacement, or by-pass and the different materials of homograft (nylon or dacron). There was a late failure rate of 14%, highest with homograft (18%) and nylon (21%) and lowest with dacron (6%). These results are apparently much better than those reported in this paper, in which only half the grafts remain patent. However, it is likely that the results of De Bakey *et alii* are weighted by a high proportion of recent grafts, and comparison is therefore not possible.

It is pertinent to ask at this point whether the operation of arterial grafting in occlusive arterial disease of the lower limbs is worth while. Less than 10% of patients with occlusive arterial disease of the lower limbs prove suitable for operation. In such cases one can, at the cost of a major surgical operation and a period of two weeks in hospital and a like period of convalescence, offer the patient an 85% chance of establishment of a functioning graft, but only about a 50% chance that the graft will still be patent in three years. The results are far from ideal, but compare favourably with various other operative procedures, such as excision

of malignant tumours, in which the over-all success rate is low. However, in many cases of occlusive arterial disease the symptom tends to remain stationary over a period of years, and the disease does not usually kill the patient. The symptomatic relief following successful operation is dramatic; in most patients with claudication, this symptom is completely abolished. Although complications of operation are infrequent, there are definite risks, particularly of infection and hæmorrhage.

In cases of election, in which the limb is not threatened with gangrene and the main symptom is claudication, the reasonable course is to put the position to the patient, who can make the decision. The operation is not advised if there is a popliteal artery of poor quality. In cases of threatened gangrene, operation is advised more strongly as providing the best means of saving the limb. However, even here there will be hesitation in advising the operation, unless there is a reasonably good distal vessel and the general condition of the patient is reasonably satisfactory. In most cases, sympathectomy will be the first mode of attack, and arterial grafting will be considered only if this fails.

Although the results of arterial grafting fall short of what was hoped for, the operation has given great relief to many patients and should be continued. The results to date give no indication for widening the scope of operation. Instead, efforts should be directed to improving technique to obtain a greater success rate, particularly in respect to graft survival. This should be combined with efforts to inhibit the progress of the atherosclerotic condition in the patient's own arteries, which may well be a factor leading to thrombosis in the graft. When, as a result of these measures, a better prognosis as to graft survival can be given, it may be justifiable to extend the scope of operation to include cases at present considered unsuitable.

Summary.

We have reviewed our experience over the past six years with arterial grafting for occlusive arterial disease of the lower limbs.

In general, operation has been performed only on patients with severe symptoms not able to be relieved by simpler methods, whose general condition was reasonably good.

Sixty-two grafts have been inserted in 52 patients by four main techniques: homograft, end-to-end; homograft, shunt; teflon prosthesis, end-to-end; teflon prosthesis shunt. The immediate success rate was 85%. In most of the failures, unfavourable factors were present before operation.

The period of graft survival has varied. Of the whole series, only 40% have survived for three years. This figure is improved to 50% by excluding the early end-to-end homografts.

Complications are rare. The most important are delayed wound healing from infection, and hæmorrhage.

The results justify the continuation of the operation, but do not warrant the extension of its use until it is possible to obtain a better prospect of graft survival.

Acknowledgements.

The first arterial graft operations in the Alfred Hospital were performed by Mr. C. J. Officer Brown. We are grateful to him for allowing us to include these cases, and for his continued guidance and encouragement in this work. We are indebted to Dr. H. A. Luke, Director, and other members of the Department of Diagnostic Radiology for the arteriographic studies.

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PNEUMATOSIS CYSTOIDES INTESTINALIS (HOMINIS).

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THE rare but interesting disease which is the subject of this paper has been described under many names: gas cysts of the intestine; intestinal emphysema; peritoneal pneumatosis; cystic lymphopneumatosis; bullous emphysema; multiple cysts of the intestine; and various others. However, pneumatosis intestinalis is its most widely recognized name.

The condition was first described in man by Du Vernoi in 1730, but the first detailed description is attributed to Bang (1876). In all, only some 300 cases have so far been reported (Calne, 1959). The condition is supposedly similar to gas cysts, found rarely in the mucosa and submucosa of the vagina and even in the bladder of pregnant women, and to the intestinal gas cysts commonly found in animals, particularly hogs, but also in sheep and chickens.

The disease may be found in man from infancy to old age, but is found mostly during middle age, being three and a half times commoner in males than in females. Mackenzie (1951), who reported 12 cases in infants and reviewed 32 pædiatric cases, found that in children the disease occurs in association with very poor general health, often with congenital or acquired disease of the gastro-intestinal tract, as well as respiratory disease and diarrhœa. He considered the condition as unimportant, and stated that in no case reported in the literature was pneumatosis held responsible for the death of a child or an infant.

The gas cysts are predominantly mucosal and submucosal in infants and subserosal in adults. The cysts vary in diameter from a few millimetres to several centimetres and are found mostly throughout the small bowel and rarely in the stomach, the mesentery, the omentum and large bowel; they are particularly uncommon in the descending and sigmoid colon. The rectum, duodenum and appendix are said to be free of the disease.

The areas involved may be localized or very extensive. The odourless, non-inflammable gas found within the cysts is made up of 90% nitrogen, 7·5% carbon dioxide and 2·5% oxygen.

Pneumatosis intestinalis is frequently associated with other diseases of the gastro-intestinal tract, such as peptic ulceration, carcinoma of the stomach, inflammatory ulceration of the intestine, intestinal obstruction and appendicitis. Pyloric stenosis is present in 60% of all cases, and 50% of all patients have duodenal or gastric ulceration. In the cases associated with stenotic duodenal ulceration, the upper part of the jejunum usually contains most of the gas cysts (Koss, 1952).

Although the condition is frequently symptomless and may be found accidentally at operation or autopsy, it may, on the other hand, cause complications such as intestinal obstruction, volvulus of the intestine, spontaneous pneumoperitoneum, intussusception and even perforation of the intestine. Diarrhœa is the most common symptom, and the passage of blood and mucus per rectum may be found when the large bowel is involved. Yunich and Fradkin (1958) have reported a case of fatal sprue secondary to extensive pneumatosis intestinalis.

Gas cysts may appear quite quickly. They have been observed to appear in a five-week interval; they may also disappear spontaneously in a relatively short period; on the other hand, they have been known to extend or increase in size or remain unchanged for prolonged periods.

Pathological Basis.

The gross pathological findings depend on the severity and site of the disease. Two types of cysts are distinguished. The serosal cysts involving the serosal surface of the affected bowel present a soap-bubble or hydatidiform appearance. The submucosal cysts are not visible on external examination of the bowel, but give the affected bowel a spongy, cushion-like consistency. When the bowel is opened, the "cobble-stone" appearance of the mucosa is seen (Figure I). In sections of the involved bowel wall, the cysts produce a honeycomb appearance (Figure II). During manipulation of the bowel, a distinct "pop" may be heard when one of the cysts is ruptured. The cysts are usually found in clusters, but they do not communicate with each other.

Microscopic examination reveals the intestinal mucosa to be normal and unbroken, the gas being contained in sacs, mostly with no well-defined lining membrane; but some cysts may be lined by a flattened or cuboidal epithelium and often giant cells. It appears that in the early stages the cysts are lined with endothelial cells, which later coalesce and form multinucleated giant cells. In time these giant cells are sloughed off, the cysts being left without any lining (Kreeger and Littman, 1954).

There is also a non-specific inflammatory reaction, which is probably a foreign-body reaction to the presence of gas.

Diagnosis.

The diagnosis of the condition depends largely on its site along the gastro-intestinal tract. In the condition in the large bowel, the diagnosis rests on the plain and barium sulphate enema X-ray studies and on the sigmoidoscopic appearances. In a number of the reported cases the similarity of the X-ray and sigmoidoscopic findings has led to the erroneous diagnosis of multiple polyposis. Even after biopsy through the sigmoidoscope and histological examination, the erroneous diagnosis has been made. However, the condition is being more frequently diagnosed before operation because of the more general awareness of its occurrence.

In the large bowel it can also be mistaken for neoplasm and advanced ulcerative colitis.

In plain scout X-ray films, gas cysts are seen as radiotranslucent areas localized to the bowel wall, which must be differentiated from gas within the lumen of the bowel. After introduction of a contrast medium, the radiotranslucency can be more readily recognized (Figure IV).

Through the sigmoidoscope the lesions when seen appear as hemispherical sessile tumours with a wide base, pale red in colour and rather translucent in appearance. A few may be seen in the rectum, and large clusters may be found in the sigmoid colon. These are less fleshy in appearance than intestinal polypi and lighter in colour, and generally have a wider base. Biopsy of the lesion will deflate the cyst, and histological examination should confirm the diagnosis.

With cysts in the small bowel or mesentery, plain X-ray films may show bizarre gas collections, and a mistaken diagnosis of a perforated viscus may be made. Pneumatosis intestinalis should be considered as a possible diagnosis in any patient having free air under the diaphragm, but not having any symptoms or signs

of a perforation of the gastro-intestinal tract. The same remarks would apply to a patient suffering from spontaneous pneumoperitoneum. Pneumatosis intestinalis is the only known condition which can explain prolonged or recurrent spontaneous pneumoperitoneum.

Ætiology.

Many theories have been advanced to explain the causation of pneumatosis intestinalis, but none has proved entirely acceptable. The neoplastic and the bacterial origin theories have been disproved. There is some evidence that dietary deficiency may be a causative factor in hogs. Sixteen out of 22 pigs fed on polished rice with protein supplement of washed casein developed intestinal emphysema. Although this condition often

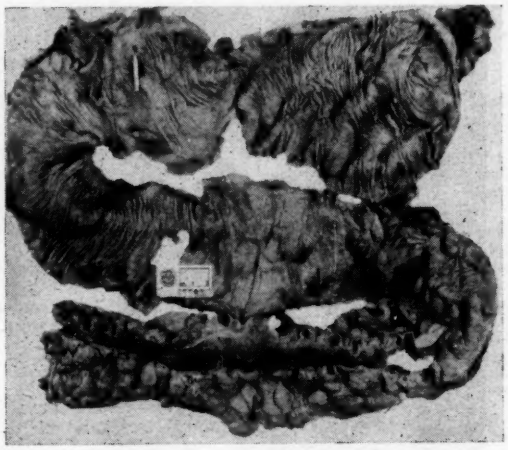

FIGURE I.

Showing the whole specimen opened. Mucosal surface. The arrow points to the opening of a diverticulum.

occurs in humans suffering from lesions of the gastro-intestinal tract interfering with a normal dietary intake, there is no strong evidence that dietary deficiency is an important factor in the causation of the human form of intestinal pneumatosis.

The mechanical theory seems to be the most acceptable explanation of the causation of the disease. Air injected directly into the gut wall follows the lymphatics to the mesentery and disappears in a few days. In intestinal pneumatosis, it appears that the intestinal gas enters through a break in the mucosa, and is forced by hyperperistalsis into the lymphatics of the bowel wall. The gas in the lymphatics causes inflammatory changes and lymphatic obstruction, with the result that the gas cannot be absorbed.

This would explain the high association of the disease in the small intestine with peptic ulceration causing pyloric obstruction, and with other gastro-intestinal lesions causing some degree of intestinal obstruction.

The gas may spread to the serosal surface via the mesentery, and so may be found at widely separated points along the bowel.

Of 213 cases reviewed (Marshak *et alii*, 1956), in only 40 was the colon affected and in only 13 was the large bowel only affected. These workers have reported three cases in which the condition followed sigmoidoscopy and biopsy or rectal surgery. In one case the pneumatosis disappeared, and repeated X-ray examinations failed to show any recurrence up to four years. In the others the disease has remained stationary.

In one case reported in this paper, the pneumatosis confined to the left half of the colon and the rectum was associated with diverticulosis. It is possible that air entered through a mucosal breach in one of these diverticula. On the other hand, the associated finding may be quite coincidental.

Thus, although the disease is usually divided into two types (the rare primary form, in which no site of entry of the gas can be discovered, and the commoner secondary type, in which other lesions of the gastro-intestinal tract are present causing the entry of gas into the lymphatics of the intestinal wall), it would perhaps be reasonable to say that in most cases careful study will reveal a breach in the mucous membrance through which the gas may enter. It is, however, difficult to explain why bacteria are not found in the gas cysts, and why peritonitis does not follow pneumoperitoneum occurring as a complication of pneumatosis intestinalis. Selective absorption of gases from the cysts may explain why the composition of the gas in the cysts is different from that of intestinal gas.

Reports of Cases.

CASE I.—Mrs. A., aged 86 years, was admitted to the Canterbury District Memorial Hospital with a history of absolute constipation, vomiting and right-sided abdominal pain of four days' duration. The patient had been in fairly good health prior to the present illness, but was suffering from mild congestive cardiac failure. She had had an appendicectomy performed about 30 years before.

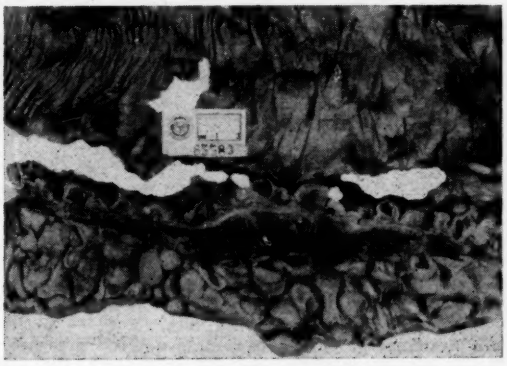

FIGURE II.

Closer view of the left colon showing cobble-stone appearance of the cysts on longitudinal section of the bowel wall.

On examination, she was in obvious abdominal pain. Her tongue was dry, but not coated. Tenderness and rigidity were present over the whole of the lower half of the abdomen, and rebound tenderness was elicited over this area. The abdomen was only slightly distended and the bowel sounds were not accentuated. On rectal examination, tenderness was elicited on the left side and the rectum was found empty. The pulse rate was regular at 90 per minute, the blood pressure was 130/90 mm. of mercury and there were signs of mild, congestive cardiac failure. The temperature was 99° F. The serum sodium content was 150 mEq/l., the serum potassium content 4·9 mEq/l. and the serum chloride content 98 mEq/l. The hæmoglobin value was 12·7 grammes per 100 ml. The leucocytes numbered 15,600 per cubic millimetre, 84% being neutrophils, 12% lymphocytes and 4% monocytes. The red cells appeared normal and platelets were plentiful. A plain X-ray film of the abdomen showed small bowel distension, but no fluid levels.

A provisional diagnosis of ruptured diverticulum of the colon or bowel obstruction was made. Laparotomy was performed through a mid-line subumbilical incision. A length of about two feet of small bowel was adherent to the appendicectomy scar. The whole of the remainder of the small bowel had undergone torsion, two complete twists in an anti-clockwise direction. A 12 inch section of the small bowel was dark and very congested, but viable. The torsion was undone and the dilated small bowel traced to the collapsed small bowel. Enormous

numbers of gas cysts were noticed on the serosal surface of the portion of the small bowel which had undergone torsion. No other abnormality was found.

After operation, the patient had considerable abdominal distension and some vomiting for 10 days, but finally made an excellent recovery, and was discharged 20 days after her admission to hospital. She died six months later of congestive cardiac failure, without developing any further gastro-intestinal symptoms.

CASE II.—D., a female patient, aged 57 years, presented with a history of loose motions, some fæcal incontinence on micturition and the passage of blood per rectum. She had a previous history of severe hypertension in 1952 (a blood pressure in the region of 260/180 mm. of mercury) and of poor renal function for 22 years. The hypertension followed toxæmia of pregnancy. She had had two minor cerebral thromboses, one in 1952 and one in 1956, and had been taking "Ansolysen" for her hyptertension for some years. Cholecystectomy had been performed for cholelithiasis in 1956; from this she had made an excellent recovery. She said that she was allergic to bromides.

FIGURE III.

Microscopic preparation showing the empty spaces almost entirely in the submucosa. There are no giant cells and there is no inflammatory reaction around these dilated lymphatics.

On examination of the patient, her blood pressure was 190/110 mm. of mercury and her pulse rate was regular at 70 per minute. Her heart was enlarged to the left. Her urine contained a considerable amount of albumin, but gave a negative result to tests for sugar. Except for her cholecystectomy scar, nothing remarkable was found on abdominal and rectal examination. X-ray examination with a barium enema produced the following findings (Figure IV):

> The enema flowed readily round to the caecum without any delay or obstruction. Numerous diverticula are scattered along the colon with here and there just a suggestion of a mild, associated diverticulitis. The main concern, however, is the extraordinary irregularity of the mucosa in the descending and sigmoid segments. In some respects the appearances suggest a form of polyposis but it is considered that there is a strong possibility of carcinoma near the mid point of the descending colon.

Sigmoidoscopic examination to 20 cm. revealed five small hemispherical sessile tumours about 5 to 10 mm. in diameter, and finely granular mucosa. There was a considerable increase both in size and in numbers of those tumours at the recto-sigmoid junction, and at 20 cm. large numbers of them could be seen in the sigmoid colon entirely replacing the bowel mucosa. Although these tumours showed the appearances typical of submucous gas cysts, they were mistaken for multiple polypi of the colon. Biopsy of these lesions was not attempted, as none appeared to have undergone malignant change, and the suspicious area seen in the barium enema X-ray examination was much higher up in the descending colon.

The patient was a poor surgical risk; but because the diagnosis of multiple polyposis with carcinoma of the sigmoid colon had been made, operation was advised. The "Ansolysen" was discontinued prior to operation.

At operation, no definite carcinoma was found; but palpation of the large bowel gave the impression that it was packed with multiple polypi, especially in the descending colon and the sigmoid colon. Total colectomy with ileo-rectal anastomosis was performed. A drainage tube was placed in the vicinity of the anastomosis. During the operation several distict "pops" could be heard whilst the large bowel was being manipulated, and only then did it occur to me that the lesions might indeed be gas cysts. Closer inspection of the serosal surface at this stage did show a few subserosal gas cysts, but it was still thought possible that these were associated with multiple polyposis of the colon.

The pathologist reported the following findings: The specimen was the greater part of the large bowel, measuring 137 cm. with 12 cm. of the terminal portion

FIGURE IV.

Radiographic appearances following barium sulphate enema. Several diverticula also present.

of the ileum attached. The appendix was absent. Except for a few (approximately six) small diverticula along the mesenteric border, the caecum, ascending colon and hepatic flexure were normal. From the right half of the transverse colon, the remainder of the bowel as far as the distal cut edge was notably and uniformly abnormal. The mucosa was thrown up into cobblestone-like, large, smooth, yellow polyps. The junction between affected and normal intestine was fairly sharp and oblique. These smooth, hemispherical polypoid protrusions of the mucosa measured from 0·5 to 2·5 cm. In a few areas there was a little intervening normal mucosa. The cut surface showed that the "polyps" consisted of empty submucosal cystic spaces with a smooth lining. There was an increase in the pericolic adipose tissue in the affected part of the bowel. A few subserosal gas cysts were also evident.

Microscopic examination (Figure III) showed that the spaces were situated almost entirely in the submucosa and that they were empty. They were lined by flattened endothelial cells. These features were strongly suggestive that the spaces were dilated lymphatics. There were no giant cells, and there was no evidence of inflammation. The mucous membrance overlying the spaces was a little atrophic, but showed no other significant abnormality. The muscularis propria was also attenuated in this region. A few small lymph nodes were found, and examination of sections showed no dilatation of lymphatic sinuses in

them. There was also no evidence of granulomatous inflammation.

After the operation, the patient was well until the fourth day, when she developed jaundice. Blood had been administered on the first and second days after the operation. The serum bilirubin content was 6·9 mg. per 100 ml., and the amount of urobilinogen in the urine was much increased, as was the amount of bile in the urine. Other liver function tests gave normal results. The blood urea content was 88 mg. per 100 ml. The temperature had risen to 101° F., and the diagnosis of pylephlebitis and portal pyæmia was made. The patient's condition became progressively worse, although her jaundice gradually faded until it had almost disappeared.

She developed severe abdominal distension, and no bowel sounds were audible for one week. Flatus and fæcal fluid motions were then passed until the day before her death. There was no drainage from the drainage tube. She died 18 days after operation. At no stage after the onset of the complication was the patient fit for reexploration.

The autopsy report was as follows:

External Appearance: Obese woman. Seven inch left paramedian incision. Drainage wound in left iliac fossa.

Cardio-Vascular System: There was a moderate pericardial effusion. Heart was slightly enlarged. Valves and coronaries normal. Moderately severe atheroma in the aorta.

Respiratory System: Moderate bilateral pleural effusion (straw-coloured). Mucopus in trachea and bronchi. Both lower lobes collapsed, showing consolidation. Upper lobes dripping with œdema.

Digestive System: General gangrenous peritonitis made detailed examination impossible. The liver was pale but normal in size and pattern. The stomach contained coffee ground matter and fluid. The region of the hepatic flexure of colon was converted into a gangrenous cavity containing fæculent fluid. The whole gut was matted together and too friable for dissection. It would appear that perforation occurred in the loop of gut occupying the space of the resected colon but no recognizable gut remained. The anastomosis in the pelvic cavity appeared to be intact.

Uro-Genital System: Both kidneys showed granular surfaces with narrowed cortices and increase in pelvic fat. The peritoneal surface of the bladder was gangrenous but the mucosa appeared normal.

Anatomical Findings:

1. Pericardial effusion.
2. Arteriosclerosis.
3. Pleural effusion.
4. Œdema and collapse of lung.
5. Hypostatic pneumonia.
6. Gangrene of gut.
7. General peritonitis.

It would appear that the peritonitis was due to perforation of the small bowel, probably following mesenteric vessel thrombosis.

Discussion.

That pneumatosis intestinalis is rare is undoubted, only some 300 cases having been so far reported. That it is frequently asymptomatic and only rarely directly the cause of death is also well established. But it may give rise to several complications of serious nature, as was stated previously, and certainly to much confusion in diagnosis, usually being mistaken for a more dangerous lesion when it occurs in the large bowel. The diagnostic confusion is mainly due to the fact that the clinician is unfamiliar with or unaware of the condition.

In Case I, the diagnosis was made at laparotomy and the pneumatosis intestinalis was of minor importance, the symptoms being due to the volvulus of the small intestine.

Case II highlights several interesting and important features of the disease when it occurs in the large bowel. The condition in this case, as in many other cases reported in the literature, was mistaken for multiple polyposis coli, so that pneumatosis intestinalis must be thought of whenever radiographic or sigmoidoscopic appearances suggest the presence of multiple polyposis in the large bowel. The differential diagnosis of the two conditions has already been discussed and it has been stressed that if the clinician, the radiologist and the pathologist are aware of the condition, the correct diagnosis is easily made when the lesions can be reached by the sigmoidoscope. Unfortunately the disease is rare in the large bowel; very rarely is it found in the sigmoid colon, and it was previously thought never to be found in the rectum. It appears that from a study of the literature available, gas cysts have been seen through the sigmoidoscope on 12 other occasions, and the rectum has been found involved in only four other cases.

The second case is therefore of interest, in that it belongs to the unusual group in which the lesions are found in the sigmoid colon and therefore are visible through a sigmoidoscope, and to the very rare group in which the lesions involve the rectum.

In many of the cases reported in which the condition involved the large bowel, the patients were asymptomatic, but among those presenting with symptoms, diarrhœa and the passage of blood and mucus per rectum were common findings. The second patient in this paper presented with the passage of blood-stained loose motions.

One unusual feature, which has not previously been reported to my knowledge, is the association of pneumatosis of the left side of the colon with diverticulosis. It is possible that the site of air entry in this case was through a breach in the mucosa in one of these diverticula. However, the finding may be purely coincidental.

Treatment.

The treatment of pneumatosis intestinalis must vary with the location and extent of the lesions, whether the condition is primary or secondary and whether it is causing symptoms or not. When the condition is secondary to obstructive lesions, such as pyloric obstruction from gastro-duodenal ulceration, relief of the obstruction is said to cure the condition. When the disease is found in the small bowel and is extensive and of the primary variety, no treatment is possible except that of the resultant complications. However, in several cases the condition has been known to decrease or even to disappear entirely. When symptoms are present and the lesions are localized, resection may be indicated.

In the large bowel, conservatism is advisable unless severe symptoms or complications, such as obstruction, are present. Several patients have been observed for years without extension of the lesion or development of complications. In the second case in the present paper, had a correct diagnosis been made, one would have been more than justified in treating the patient conservatively in spite of the extent of the cysts, the patient being a poor surgical risk.

If conservatism is to be adopted, care should be taken not to rupture or puncture the cysts because of the danger of later perforation of the bowel.

Summary.

1. Pneumatosis cystoides intestinalis is a rare condition caused by the entrance of intestinal gas into the lymphatics of the bowel wall. The gas may be mainly distributed to the subserous or submucous surfaces.

2. The disease is rarely primary—that is, it rarely occurs when no other associated gastro-intestinal lesion is present. Much more commonly it is secondary to ulcerative and/or obstructive lesions of the gastro-intestinal tract, in 60% of all cases being associated with pyloric stenosis and in 50% of all cases being associated with duodenal or gastric ulceration. In three cases it has been reported as following sigmoidoscopy and biopsy

or rectal surgery. The association of extensive pneumatosis of the left side of the colon and the rectum with diverticulosis apparently not hitherto reported is here recorded (Case II). This may or may not be of ætiological significance.

3. The condition may be found at all ages, but occurs most commonly during middle age, and is three and a half times more common in males than in females. In infants it is commonly associated with other abnormalities of the gastro-intestinal tract, and respiratory diseases and diarrhœa often coexist.

4. In many cases the condition is symptomless. Diarrhœa is the most common symptom when symptoms are present, and blood and mucus may be passed per rectum when the large bowel is involved. Intestinal obstruction, volvulus, spontaneous pneumoperitoneum, intussusception and perforation of the intestine are some of the recorded complications.

5. Although rarely found in the large bowel, pneumatosis causes much confusion in diagnosis when it is found in the left side of the colon and rectum, being commonly mistaken for multiple polyposis. There have been only 12 other cases recorded in which the lesions have been seen sigmoidoscopically, and only four other cases in which the rectum was involved.

6. When the disease involves the small bowel and is secondary to an obstructive lesion, such as pyloric obstruction, relief of the obstruction will probably cure the pneumatosis. Localized lesions giving rise to complications may require resection. Even in extensive cases, spontaneous resolution may occur. In the large bowel, conservatism is advisable unless severe symptoms or complications make resection essential.

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Reviews.

Man's Posture: Electromyographic Studies. By J. Joseph, M.D., M.R.C.O.G.; 1960. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 5¾", pp. 98, with illustrations. Price: 44s. (English).

DR. JOSEPH is an acknowledged authority on electromyography, and his work on this aspect of functional anatomy has received the general approbation of the Anatomical Society of Great Britain and Ireland. So far as subject matter is concerned, this small book of 77 pages of text is divided into three sections—(i) man's posture, (ii) technical details of electromyography, (iii) consideration of regional muscle groups in relation to posture.

The work as a whole is well thought out, and the correlation between the first and third sections is an intimate one; yet perhaps students of physical education and physiotherapy would find insufficient dogma and too much debate to satisfy their needs. However, the book is of absorbing interest to investigators of functional anatomy in general and of postural mechanics in particular, and should find a place on the bookshelves of every anatomist.

One criticism may be offered: with the increasingly coordinated teaching of anatomy and physiology, it seems a pity that Dr. Joseph has not laid more stress upon the correlation of structure with function. It would have been pleasing to have some reference to the variety of shapes of bony articular surfaces and modification of metaphyseal and epiphyseal structure. Moreover, muscle morphology, macroscopically as well as microscopically, should surely find a place in postural considerations. The myohæmoglobin content of postural muscles is of considerable interest, and indeed may have some bearing on electromyographic response.

The very generous bibliography which Dr. Joseph provides does not do justice to his own extensive investigations on this subject.

Adventure to Motherhood: The Picture Story of Pregnancy and Childbirth. By J. Allan Offen, M.D.; 1960. New York: Taplinger Publishing Co., Inc. 7¾" × 5¼", pp. 74, with many illustrations. Price: $ 2.95.

THIS is a pocket-sized gift book presenting pregnancy at its glossiest. In full-page coloured photographs we trace the story of a very attractive young American matron from the time when she "hopefully suspects" that she is pregnant to the delivery of her baby. Large coloured illustrations show clearly the baby's exact size and position at various stages, and a small amount of text explains the different examinations undergone by the delighted mother. She is pictured eating platefuls of salad, shopping at the supermarket, taking a mid-day rest, selecting maternity fashions and visiting her dentist. It is all one big romp, until life becomes earnest when she and her boyish husband are taken on an "orientation tour" of the maternity wing of the hospital. Here they are shown the delivery rooms and a great deal of impressive equipment. When labour begins, we are treated to page after page of artistic photographs of the delivery, which unfortunately is largely hidden by

the mother's abdomen and the anæsthetic machine, and is performed under full anæsthesia with forceps. Then the baby, wringing wet and covered with blood, is shown to the happy father, who rushes out to telephone the relatives.

It is all so intensely American, with the vitamin pills, beauty preparations, packaged milk, orientation tours and, one suspects, the forceps, that one feels rather like a reader of the latest woman's magazine from that country. Those whose morale is boosted by this sort of thing will enjoy this book; but we think that it is a grave mistake to present, in a book designed to reassure the mother-to-be, a mode of delivery which we in Australia regard as quite abnormal.

Annual Review of Medicine. Edited by David A. Rytand and assisted by William P. Creger; Vol. 11, 1960. Palo Alto, California: Annual Reviews, Inc. 8½" × 6", pp. 464. Price: $7.50.

THE object of this kind of book is to present a summary of the most recent developments in a scientific field without being merely an extensive bibliography. Since some thirty different experts have contributed to this volume, such an object is achieved in varying degrees by different authors. However, as usual, this annual review is readable as well as informative and, of course, wholeheartedly up to date.

It mirrors the trend of our age towards specialization, with detailed discussions of subjects of limited interest such as high-voltage radiation therapy, the L.E. cell phenomenon and homograft sensitivity. In this category, too, we find comprehensive sermons on acute renal failure, the perinatal circulation and tissue and host specificity in viral disease.

Of more general interest are chapters dealing with arterial, pulmonary and portal hypertension, thyroid disease and physiological aspects of diuretics. Most readable and topical of all the articles is a study of behavioural problems in the adolescent, and perhaps most intriguing, the contribution describing hormonal control of pigmentation and the balance between melanocyte stimulating hormone (MSH) of the pituitary and melatonin, a lightening substance found in the pineal body and peripheral nerves.

As usual, the fields of bacterial disease, neoplasia, hæmatology, pædiatrics, dermatology and gastro-enterology all receive attention, reference being made to the most recent work done in each specialty.

It is noteworthy that two or three overseas contributors figure among the authors, indicating the growing international flavour of this series.

Radiation: Use and Control in Industrial Application. By Charles Wesley Shilling, M.D., Sc.D.; 1960. New York and London: Grune & Stratton. 8¼" × 5¼", pp. 232. Price: $6.75.

THIS book was written as one in a series of modern monographs in industrial medicine with the object of preparing the industrial physician for his entry into the field of radiation hazards. It is a compact little book of two hundred pages, which deals generally and, of necessity, superficially with radiobiology, atomic medicine, radiological health practices and some of the facts concerning atomic power and the industrial use of isotopes. Written by the Deputy Director of the Division of Biology and Medicine of the United States Atomic Energy Commission, it gives an authoritative and informed coverage of diverse matters, which can be grouped only under a title as broad as "Radiation". It could also fall into the category of a publication for undergraduates and the medical practitioner at the general-information level, rather than for the specialist radiologist.

The first section discusses simply, in 25 pages, radiation and nuclear physics applying the discussion to consideration of the origin of background radiation to which we are all subject. The effects of radiation over and above this natural background are given briefly in the next section of 50 pages. It includes biological measurements, and a well-balanced view of the genetic effects is presented. Prevention and treatment of some of the effects discussed in the previous section follow in the third section, again of some 50 pages. The principles are well conveyed by means of diagrams and tabulations. Monitoring, decontamination and waste disposal follow in another section of equal length, together with personnel exposure limits, which are explained with the concept of the maximum permissible dose sensibly stated. Radioisotopes are too transiently described in the last section of about 30 pages to give more than a passing acquaintance with their derivation and application.

The irritating features of the book are the typographical errors, and the fact that it does not give as a reference (some sixty major references are listed at the back) any work originating outside the United States of America. The monograph would seem big enough to overcome this smallness of outlook.

Symposium on Salt and Water Metabolism. Edited by Alfred P. Fishman, M.D.; Circulation, Vol. 21, No. 5, Part Two, Supplement, May, 1960. New York City: American Heart Association; New York Heart Association. 11" × 7½", pp. 803-1060. Price: $2.00.

HERE is an exciting and informative collection of papers, together with a transcript of the discussion they aroused, giving an insight into the ways in which scientists are thinking and tackling the many problems of salt and water metabolism and some indication of their achievements. It is obvious that "The participants shared a lively interest in salt and water metabolism", as the Guest Editor, Alfred P. Fishman, writes, "but differed considerably in their scientific orientations: they included physical chemists, physiologists, zoologists, and physicians; their topics ranged from the physical chemistry of solutions to the transport of electrolytes and water across biological membranes".

Thus there are accounts, and pictures, of the physical structure and behaviour of water and electrolytes in solution, of protein molecules, and of the physical properties of membranes across which these materials have to be transported. Robert Pitts introduces the section dealing with renal mechanisms, in which the subjects include osmotic concentration and dilution in the nephron, and the function of the collecting ducts in the transport of water and tubular activities in the handling of ions, including results obtained by the stop-flow technique. Lessons from comparative biology have emphasized the value of studying the biochemical changes associated with anatomical metamorphosis in the vertebrates. Then there is a series of papers dealing with extrarenal regulation of salt and water, roving over such varied topics as ion transport in the gut, in the eye and in bile, the salt-secreting gland of marine birds, volume receptors, antidiuretic hormones and so on.

This volume will be avidly perused by all who thirst after knowledge in this field, and many others will find enjoyment by thumbing its pages to glimpse and marvel at the complexities which underlie our everyday biological processes and their gradual elucidation.

The Management of the Doctor-Patient Relationship. By Richard H. Blum, Ph.D.; 1960. New York, Toronto and London: McGraw-Hill Book Company, Inc. 9" × 6", pp. 320. Price: $8.50.

ANYONE who thinks about the nature of his work as a medical man can become quite depressed when he considers the impact on his calling of technology, specialism, urbanization (as opposed to civilization) and the growth of the mass-media of communication. A case could even be made out for the existence of a doctor-patient barrier, which is sometimes found instead of the *rapport* on which psychiatrists, at any rate, insist. Neither doctors nor patients are what they were in the "good old days", when the doctor's strong silent bedside manner was a cloak for his ignorance, and he could commit almost any clinical or therapeutic crime upon his innocent patient and get away with it. Now patients are not innocent, thanks to Paul de Kruif and George Sava, *The Reader's Digest* and "Young Doctor Kildare"; and doctors, we hope, are not ignorant. However, most doctors probably are ignorant of the possible variations in the patient's perspective of health, sickness and medical care. The undergraduate course, and an increasingly long period of hospital residencies after graduation, ensure that a doctor will have buried his roots so deep in the soil of medical science by the time he first treats a patient single-handed that his ability to grasp the nuances of life in the rest of the world will be seriously restricted.

This book, by a psychologist, not a medical graduate, is designed to improve the practising doctor's comprehension of life in the rest of the world, and of his place in this world. It succeeds completely in achieving its objective, which is to guide physicians in their management of the doctor-patient relationship. It is comprehensive, systematic, learned, witty, even funny in places as *Punch* or *The New Yorker* is funny. Most of what matters about the doctor-patient relationship is clearly stated. There are full bibliographies at the end of each chapter, and it is good to find an American writer so widely acquainted with British as well as American literature—he quotes Balint, Bowlby and Richard Asher, for example, although he misspells the name of the late Sir Clifford Allbutt.

The only serious fault is a failure to discuss adequately the role of the clinician in health education. There is quite a long chapter on "Patient Ignorance and its Cure", but only a small part of this is devoted specifically to health education, and this only in a limited way. By inference the subject is mentioned elsewhere from time to time; but nowhere is there a discussion of health counselling, marriage guidance and vocational counselling as functions of the doctor. Nor is there any mention of the "catalytic" role of the family doctor in the developing field of preventive psychiatry, where tactful intervention can sometimes correct disturbances in the parent-child relationship. One might have hoped for consideration of this at length in such a book, particularly by a psychologist.

Such cultural differences as exist between the United States of America and Australia do not, with trifling exceptions, influence the application of the author's argument to conditions in this country.

This book can be recommended to all medical practitioners—general practitioners, physicians, and psychiatrists with leisure to savour it will find it delightful; and it ought to be compulsory reading for those whose inclinations or scope of practice expose them to the risk of forgetting that patients are people.

Handbook for School Medical Officers. Edited by H. I. Robinson, M.D.; 1960. Victoria: School Medical Service, Department of Health. 8¼" × 5½", pp. 144. Price: 10s.

IT is interesting to speculate upon the fate of the present flood of medical works at the hands of some future medical historian. It may well be that the more pretentious and ponderous will be "shelved" in a special section devoted to Plagiarius; in which case the smaller ones, rooted in actual day-to-day practice, may be found of greater interest in the end.

The volume under review represents the combined experience of a team of family doctors who, in their later years, have passed into a service which examines more than 100,000 children each year. The type of examination differs from that employed by the general practitioner, whose experience is confined almost entirely to the sick; however, those who examine for entry into holiday camps, those few who act as doctors to private schools and those (fewer still) who administer routine health checks to their young patients will find much to interest and enlighten them.

The present trend in preventive pædiatrics towards integrating the work of the general practitioner and that of the medical officer of the health department may be hastened and made more rewarding for all by a study of this book. A school medical officer will find in its pages succinct answers to most of the queries arising from his work. The all-important subjects of sight, hearing and speech are very well covered, while the chapters dealing with the heart, anæmia, orthopædic defects and disfigurements are especially good.

The book admirably fulfils its purpose.

Electroencephalography in Anesthesiology. By Albert Faulconer, Jr., M.D., M.S., and Reginald G. Bickford, M.B., Ch.B., M.R.C.P.; 1960. Springfield, Illinois: Charles C. Thomas, Oxford: Blackwell Scientific Publications. 9" × 5¾", pp. 98, with illustrations. Price: 38s. (English).

To the average British reader, the words "anæsthesia" and "anæsthesiology" are synonymous. However, in the publication under review, "anæsthesiology" is used with the North American spelling and meaning. The book describes not only the changes produced in the electroencephalogram by various anæsthetic agents, but also the effects of other factors that may vary during the course of an anæsthetic administration.

Much of our present knowledge of this subject has come from work done at the Mayo Clinic. Accordingly, since the joint authors are members of that clinic, it is not surprising to find this book covering the field in a knowledgeable manner. No extravagant claims are made for the value of the electroencephalogram used during anæsthesia, but the practical applications of the method are clearly and briefly stated in the final paragraph of Chapter I. The chapters which follow then elaborate this introduction.

Generally speaking, the book avoids the technical terminology of electroencephalography. The authors prefer to refer to frequencies in cycles per second rather than as alpha, beta, delta, etc. However, at times they lapse into the latter form without explanation of the frequencies involved. To the reader not conversant with the rhythms, this may be confusing.

Chapter 2 deals with the mechanism of electroencephalographic changes and shows how little is known of the subject. Chapter 3 deals with instrumentation, and it is immediately evident that the writers are fully aware of the practical problems involved. Four chapters set out the changes produced by the administration of various anæsthetic agents and by patho-physiological changes occurring during anæsthesia.

The final chapter covers servo-anæsthesia, whereby automatic control of anæsthetic depth is established by the use of potentials derived from the electroencephalogram.

The book concludes with a very adequate bibliography, not only of American literature, but also of British, European and Australian contributions to the subject.

Altogether, this publication presents today's knowledge of the use of electroencephalography during anæsthesia in a satisfactory manner. It will provide the newcomer to the field with a basis upon which to build and direction as to where he can obtain further information.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Lectures on Hæmatology", edited by F. G. J. Hayhoe, M.A., M.D., M.R.C.P.; 1960. London: Cambridge University Press. 9¾" × 6", pp. 260, with illustrations. Price: 60s. net (English).

"Clinical Anatomy: A Revision and Applied Anatomy for Clinical Students", by Harold Ellis, M.A., M.Ch. (Oxon.), F.R.C.S.; 1960. Oxford: Blackwell Scientific Publications. 8¾" × 5½", pp. 386, with illustrations. Price: 37s. 6d. (English).

"Facing Retirement: A Guide to the Middle Aged and Elderly", by A Country Doctor, M.A., M.D., with a foreword by J. W. Robertson Scott, C.H., M.A. (Hon.), Oxon.; 1960. London: George Allen & Unwin Ltd. 8½" × 5½", pp. 128. Price: 12s. 6d. (English).

"Uprooting and Resettlement", World Federation for Mental Health; Eleventh Annual Meeting of the World Federation for Mental Health, Vienna, Austria, August, 1958; 1960. London: H. K. Lewis & Co. Ltd. 8½" × 6", pp. 150. Price: 10s. 0d. net (English).

"Clinical Child Psychiatry", by Kenneth Soddy, M.D., D.P.M.; 1960. London: Baillière, Tindall and Cox. 9¼" × 6", pp. 480. Price: 42s. (English).

"The Practitioner's Handbook", edited by William A. R. Thomson, M.D.; 1960. London: Cassell & Company Ltd. 9¾" × 7¼", pp. 734. Price: 63s. net (English).

"Meaning and Methods of Diagnosis in Clinical Psychiatry", by Thomas A. Loftus, M.D.; 1960. Philadelphia: Lea & Febiger, Sydney: Angus & Robertson Ltd. 9¼" × 5¾", pp. 170. Price: 55s.

"The Anonymous Mycobacteria in Human Disease", edited by John S. Chapman, M.D.; The Sixth Annual Ivan H. Mattson I Memorial Conference, of The University of Texas Southwestern Medical School and Dallas Tuberculosis Association; 1960. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications Ltd. 9" × 6", pp. 184, with illustrations. Price: 60s. (English).

"Transactions of the International Society of Plastic Surgeons", edited by A. B. Wallace, M.Sc., F.R.C.S.Ed.; Second Congress, London, 1959; 1960. Edinburgh and London: E. & S. Livingstone Ltd. 10" × 6½", pp. 614, with many illustrations. Price: £4 10s. net (English).

"Genetics: Genetic Information and the Control of Protein Structure and Function", edited by H. Eldon Sutton, Ph.D.; Transactions of the First Conference, October 19, 20, 21, and 22, 1959, Princeton, N.J.; 1960. New York Josiah Macy, Jr. Foundation. 9" × 6", pp. 230. Price: $6.00.

"The Croonian Lectures: delivered at the Royal College of Physicians, London in 1849: On Some of the Forms of Insanity", by John Conolly, M.D., D.C.L.; 1960. Southall, Middlesex: St. Bernard's Hospital Management Committee. 9¼" × 6", pp. 98. Price: Not stated.

"Lipide Metabolism", edited by Konrad Bloch; 1960. New York and London: John Wiley & Sons, Inc. 9" × 6", pp. 430. Price: not stated.

"Guide to the Study of the Anatomy of the Shark, Necturus, and the Cat", by Samuel Eddy, *et alii*; third edition; 1960. New York and London: John Wiley & Sons, Inc. 9" × 6", pp. 158, with illustrations. Price: $3.50.

The Medical Journal of Australia

SATURDAY, JANUARY 7, 1961.

TREATMENT OF SERIOUS INFECTIONS.

IT has become somewhat of a *cliché* nowadays to affirm that we are living in a "Golden Age" of antibiotic therapy, and it is undoubted that these agents have saved hundreds of thousands of lives in the past twenty-five years. There is, however, another side to this glowing picture, which is being slowly and perhaps reluctantly pieced together as evidence accumulates that more and more bacteria are escaping from the thrall of the "wonder drugs". At times, this subject becomes a plaything for the Press, as shown for instance in recent sensational journalism relating to the depredations of the "golden staph". But there is a growing output of communications from those qualified by experience and authority to judge the present position which demonstrates that the antibiotic tide has turned, and ground is now being steadily lost.

A lecture recently given in Boston by a recognized leader in antibiotic research, Dr. Maxwell Finland, points to the need for a "new look" at the problem of the prevention and treatment of serious infections. For his purpose, he has taken from the records of the Boston City Hospital figures relating to the relative frequency of occurrence of major pathogens in all patients with bacteriæmia, and the number of deaths therefrom, in the years from 1935 to 1957 inclusive. The most striking contrasts, of course, are provided by comparison of the results[1] in the years before the introduction of modern antibacterial agents with those of the present period. Some of the findings can only be described as startling, and at first sight might be regarded as possibly peculiar to this particular hospital. It is admitted that intensive research into blood-borne infections has been a special project here for many years, but there seems no reason to doubt that similar figures could be obtained from hospitals in other parts of the world, not excluding Australia. Further, earlier papers from Finland's pen have revealed quite clearly the trends he now describes, and they have not been challenged.

Very briefly, over-all figures for the occurrence and mortality of bacteriæmia at the Boston City Hospital showed a sharp increase in the number of cases from 1935 to 1941 (when the major sulphonamides were in use), but a slight drop in the number of deaths; this gave a fall in case fatality rate from 57·6% to 36·6%. The figure fell further to 30·0% in 1947, when penicillin was fully available. Since then, there has been a slow but steady rise in mortality, reaching 38·1% in 1957. The welter of new and broad-spectrum antibiotics has made no impression. This is really a surprising and alarming fact.

Further analysis is extremely interesting and reveals just what has happened. Here we cannot do better than quote from the author's discussion:

> . . . it appears that, whereas pneumococci and hæmolytic streptococci were formerly the most common causes of bacteremic infections and accounted for more than half the cases and nearly three fourths of all deaths from such infections, they now account for less than a fifth of the cases and less than an eighth of the deaths. In sharp contrast are the changes in *Staph. aureus* and the organisms that have generally been considered normal constituents of the bowel flora—namely, *Esch. coli, A. aerogenes,* proteus, pseudomonas and enterococcus—which may be lumped together under the term enterobacteria only for brevity and convenience, although it may not be taxonomically precise. In 1935 *Staph. aureus* accounted for slightly more than 1 of every 5 cases and less than 1 of every 5 deaths from bacteremic infections, whereas in 1957 it accounted for about two fifths of both the cases and the deaths. What are here called normal enterobacteria accounted for less than 1 in 8 cases and 1 in 11 deaths before the chemotherapeutic era; now they account for over a third of all the cases and two fifths of the deaths from bacteremic infections. . . . In other words the bacteria that were the most important causes of severe infection and mortality and were also the most susceptible to modern antibacterial agents have been reduced to a relatively minor role, whereas those that were much less frequent or extremely rare causes of bacteremia before 1935 and are also the least influenced by these drugs have much more than compensated for the difference both in the numbers of cases and in the numbers of deaths. . . . these few bacteria have been responsible for all but a small proportion of bacteremic infections and nearly all deaths from these infections.

Finland offers a few comments on this deplorable situation, blaming in large measure "the excessive and almost universal resort to antimicrobial agents, particularly in the hope of preventing unknown or ill-defined infection". Laxity in asepsis, the incidence of cross infections, the increasing average age of hospital patients and the frequency of chronic urinary tract infections (especially with enterobacterial flora) are among the other possible causes he cites. He records that no "remedies" have so far proved of avail, but urges "general recognition of the futility of chemoprophylaxis directed at preventing infections in general, and particularly in clean wounds, in newborn infants, in acute viral respiratory infections, in the urinary tract of patients subjected to indwelling catheters and in many other situations", and the realization that such usage is mainly responsible for the increase in bacterial resistance. This warning has been given before, many times, and reference may be made here to the recent article on the subject in the "Brush up Your Medicine" series in this journal.[2]

Is the lesson being heeded? Perhaps the message should be attractively printed on a stout card suitable for hanging in the doctor's consulting room, or better still in his waiting room, where it might do more good, by damping down the importunate demands (real or fancied) of

[1] *New Engl. J. Med.*, 1960, 263: 207 (August 4).

[2] MED. J. AUST., 1960, 2: 792 (November 12).

patients and their friends to have the "benefits" [*sic*] of the "life-saving" drugs. Possibly Australian hospitals can show better figures than those quoted by Finland; at least it would be interesting to have some comparison, provided adequate techniques were available for the not-so-simple diagnosis of bacteriæmia. Meanwhile, the findings of Finland's studies cannot but be a source of concern to all who are interested in the hospital management of severe infections.

SIR HOWARD FLOREY, P.R.S.

SIR HOWARD FLOREY has been elected President of the Royal Society.[1] This is a very high honour, and we know that we speak for the whole medical profession in Australia when we offer warm congratulations to this distinguished Australian. A native of the city of Adelaide and a graduate of its university, Sir Howard Florey has made his career in England since he went to Oxford as a Rhodes Scholar in 1921. However, he has maintained close contact with Australia, and in particular, on the invitation of the Commonwealth Government, played a major part in the planning of the John Curtin School of Medical Research at the Australian National University. It was hoped at one stage that he would accept permanent leadership in the work of that school, but he chose otherwise and has continued in his position as professor of pathology at Oxford, the chair which he has held since 1935. On his contribution to science and medicine it is not necessary to enlarge at present beyond pointing out that he has done many things of importance in addition to his work on penicillin, which has naturally attracted most notice both inside and outside the world of medicine because of its revolutionary effect upon medical treatment.

Sir Howard Florey's election to presidential office is particularly gratifying at this time of the Royal Society's tercentenary celebrations. Referring to these recently,[2] we pointed out the association with Australia of one of the Royal Society's outstanding presidents of an earlier day, Sir Joseph Banks. Up till then no Australian had held the office of president, and it will give no little pleasure to the members of the medical profession in Australia to learn that that honour has gone to one of their colleagues. It will be a source of justifiable delight to the Medical School of the University of Adelaide, particularly on top of the fact that Adelaide has produced three of the six Australian medical men who are Fellows of the Royal Society (the other three are Melbourne graduates). Sir Howard Florey, by his attainment of this personally deserved distinction, brings honour to his native city and to his native land. We offer him our congratulations and thanks.

Current Comment.

"VOICE OF MEDICINE."

SOMETHING quite new in the field of medical journalism has arrived in *Voice of Medicine*, which is described as "The Recorded Medical Journal". It has been prepared under the direction and supervision of the Excerpta Medica Foundation, with a board of chief editors consisting of Martinus W. Woerdeman (Amsterdam), Morris Fishbein (Chicago) and Sir Heneage Ogilvie (London). Each number consists of a series of on-the-spot recordings on a long-playing microgroove unbreakable record (hi-fidelity recording, 33⅓ r.p.m.). This is enclosed in an attractive cardboard case with a printed synopsis. Volume 1, No. 1, April-May, 1960, has as its special feature recorded reports from the Conference of the International Study Center for Rheumatic Diseases, in the form of a symposium; this was recorded in Amsterdam. A person-to-person feature follows, in which the editors interview Professor V. P. Demikhov in the Sechenov Institute, Moscow, on the subject of "The Transplantation of Organs"; this was recorded in Moscow. An every-day practice panel discussion deals with "Your Ulcer Patient", with Morris Fishbein as moderator and Lester R. Dragstedt, Joseph B. Kirsner, Philip Thorek and Asher Winkelstein as panel members; this was recorded in New York. A selection of recorded abstracts from the world's current medical literature completes the number. Volume 1, No. 2, June-July, 1960, contains as its special feature a report on ulcerative colitis from the International Congress of Gastroenterology in the form of a symposium recorded in Leyden, the Netherlands. The editors interview Professor C. G. Rob and T. Holmes Sellors on "Open Heart Surgery" in London, and a panel in New York discuss "The Use and Abuse of Psychic Energizers". The recordings are all excellent, their effectiveness being increased if anything by occasional background noises, and the personality of the contributors makes a strong impact in a way not possible in written journals. The exclusive distributors of *Voice of Medicine* are Recordo-Med Inc., 100 Central Park, South, New York 19, N.Y., U.S.A.

MODIFIABILITY OF THE NEURON.

THE astonishing specificity of nerve cells has lately been emphasized by Paul Weiss.[1] More and more refined techniques during 38 years have confirmed his earliest speculations. But the latest developments of the tripartite methodology (optical, electrical and chemical) have proved sadly inadequate to probe the mysteries of cell specificity. And the workers in each method are limited by the restricted vocabulary of their technique. We still think erroneously, Weiss states, of neurons as "live" in a sense not much different from that applied to a telegraph wire, and we think of them as all of one kind and character and static throughout life. Anatomists think of nerve cells as matured, and the changes in the process of maturation are seldom discussed. Nerve cells do not reproduce by mitosis, but each one is in a continuous state of flux and self-renewal. The tips of the finest branches of the nerve are continually dying and being renewed; new axoplasm is being generated in the cell body and streams down the axon to its tip.

Weiss believes that neuronal specificity is of two kinds: either cell A imposes its own specificity on a more plastic cell B; or if A and B are already fixed in specificities, their degree of affinity will determine whether or not they will become linked. Since 1922 he has been showing that individual muscles and individual receptor areas have distinctive qualities which they confer on the nerve cells to which they are attached during development. By this the neurons are modulated and have a new specificity imposed on them. These neurons in their turn impose a similar specificity in the next cells in an ascending scale. Thus nerve centres are briefed on what central stations are connected with each muscle or receptor (proprioceptor, cornea, retina, skin, etc.). Subtle selective linkages are necessary for the orderly functioning of the nervous system. Specifically matching properties comprise a basic principle of neural intercommunication. Neuronal specificity can be modified by terminal effectors and receptors,

[1] *Brit. med. J.*, 1960, 2: 1722 (December 10).
[2] MED. J. AUST., 1960, 2: 865 (November 26).

[1] *A.M.A. Arch. Neurol.*, 1960, 2: 595 (June).

and by virtue of this acquired specificity can selectively enter into a higher order—a union of concordant elements.

Neurons share these properties, according to Weiss, with other living cells, which display them in reproduction, differentiation, growth control and antibody production. Cells recognize each other's kinds on contact and react discriminately, remaining associated if they are alike, but separating again if they differ. Once recognized, the specificity of interrelationship between nerve cells and body cells presents us with new questions. The plasticity of the nervous system depends on the adaptive modifiability of the neurons. The stream which flows down the axon replenishes degraded macromolecules at the tip of the nerve, and serves as a vehicle for neurohumours. Weiss believes that a revision of the teaching of neurology is necessary; the new fact of the additional freedom of the neuron will have to be taken into account.

SMALLPOX PRECAUTIONS.

OVERSEAS passengers reaching Australia by air who cannot produce satisfactory evidence that they have been vaccinated against smallpox must be quarantined for 14 days from the date of their departure unless they accept vaccination. This instruction has been issued by the Commonwealth Department of Health to quarantine officers throughout Australia. The Commonwealth Minister for Health, Dr. D. A. Cameron, announced recently that every person aged more than 12 months would be required either to present a satisfactory certificate of vaccination within the past three years, or accept vaccination, unless he or she could exhibit clinical signs acceptable to the quarantine officer of having undergone vaccination within three years. A person whose vaccination certificate was apparently quite genuine but was unsatisfactory for some minor technical reason would be permitted to leave the airport, but would be kept under medical surveillance for the prescribed period. Dr. Cameron said that the exception was made for infants because a strong body of medical opinion overseas considered that vaccination of babies was potentially injurious. It was felt, also, that there was little risk of the infant of vaccinated parents spreading the disease. Vaccination was not required of passengers from New Zealand, New Guinea or the nearby Pacific Islands, where there was no smallpox.

As Dr. Cameron has said, there can be no question of the need for these precautions. If the point is not sufficiently obvious to everyone, it is brought home by the recent instances, quoted by Dr. Cameron, in which smallpox infection was carried from one country to another by air. In West Germany early in 1959 a doctor returning from India caused an outbreak of 18 cases, of which two ended in death. An infection brought in from Yemen caused 122 cases, with 33 deaths, in Aden Protectorate. These cases, and a recent incident in Britain, strongly impress the need for effective immunization by regular vaccination, especially on the part of hospital, medical and airport personnel. It is pointed out that the Commonwealth makes vaccine available free, at all times, to every person considered to be "at risk".

THE FIRST HUNDRED YEARS OF ANÆSTHESIA.

IT seems to have have taken the medical profession a very long time to feel a stirring of its conscience concerning the untoward pharmacological and psychosomatic reactions of some of their patients ever since the benefits of general anæsthesia were first made available to them in 1846. Not so long ago, a city coroner of shrewd and critical judgement told a friend in confidence that he was not entirely convinced by medical evidence that some anæsthetic deaths were caused by a persistence of the thymus gland and by the onset of a fatal reaction commonly known as "status lymphaticus". The misconceptions, flounderings and tragic mistakes so frequent in past anæsthetic practice because of the too casual approach to a subject hitherto regarded as of "minor importance" are candidly and humorously discussed in the first volume of essays by Dr. W. Stanley Sykes.[1] This writer began his career as a general practitioner and then spent 25 years as a specialist anæsthetist in Leeds, before retiring after the second World War to prepare a full history of anæsthesia, to be illustrated by representations of every conceivable kind of apparatus designed by human ingenuity or stupidity during the period under review.

Throughout the whole text, Dr. Sykes expresses himself in a whimsical and uninhibited style peculiarly his own; his communication with the reader is intimately personal, and not lacking in amusing and caustic comments on professional dealings with the new anæsthetic drugs in the early stages of their development in surgical and midwifery practice. The opening chapters are remarkable for a discursive propensity and for several lengthy digressions on topics which seem rather remote from the real subject in hand. Nevertheless, in spite of an unconventional presentation of historical facts, it is evident that the author has gone to no end of trouble to gather material from every available source, so that the book contains much information not previously recorded by the many historians of this fascinating subject.

THE EPIDEMIOLOGY OF POLIOMYELITIS.

MANY problems in the epidemiology of poliomyelitis are still unsolved. It has been noted, for example, that in countries with primitive hygiene, antibodies appear at an early age, but there is little overt paralysis. Also the age of patients with paralysis is higher in the countries with good hygiene than it used to be. A. Lindahl[2] has set out to compare various social features of the paralytic cases of poliomyelitis occurring in the Copenhagen epidemic of 1952 with those in a control series of the general population. It is well known that the paralytic cases form only a small proportion of those in which there is infection with the virus. Lindahl has noticed that rarely was the paralytic case in the youngest member in the family, and only rarely were adults paralysed who belonged to a household with no small child. He finds that in the households in which the paralysed cases occur, there are children aged one to five years more frequently than in control houses. He believes that often such children are the first to be infected in the family and that they excrete poliomyelitis virus. Other members in the household then receive a massive dose of the virus. In poliomyelitis, as in many diseases, it is believed that a massive initial dose of the virus is more likely to cause serious disease. Some contributory evidence is that the children aged one to five years appear to have had their disease epidemic about a week earlier than those at other ages. Lindahl believes that if an older child or parent is the first in the family to be infected, the other members are not likely to be subjected to such massive doses because the personal hygiene of the older members is relatively good. Further, there is little chance of being able to limit the spread of the disease by quarantine, but possibly some good would come in epidemic times from warning families that the danger of contracting a massive dose of virus from a small child is not negligible.

[1] "Essays on the First Hundred Years of Anæsthesia", by W. Stanley Sykes, M.B.E., B.Chir. (Cantab.), D.A.; Volume I; 1960. Edinburgh: E. & S. Livingstone Ltd. 8¾" × 6", pp. 174 with many illustrations. Price: 30s. net (English).

[2] "The Poliomyelitis Outbreak in Copenhagen in 1952: Epidemiological Studies", by Alfred Lindahl; *Acta medica scandinavica*, Supplementum 355; 1960. Copenhagen: Acta medica Scandinavica. 9¼" × 6¾", pp. 168. Price: Subscription.

Abstracts from Medical Literature.

DERMATOLOGY.

Effects of Prolonged Administration of Griseofulvin.

C. S. LUNGOOD *et alii* (*A.M.A. Arch. Derm.*, May, 1960) report the effects of prolonged griseofulvin administration on liver, hæmatopoietic system and kidneys. Griseofulvin was given to 66 patients with superficial fungous infections and the patients were carefully observed for side effects and signs of toxicity. In adults the dosage was 280 mg. four times a day and in children 125 mg. to 250 mg. twice daily, depending on the age and weight of the patient. There were no significant changes in any of the cellular elements of the peripheral blood. Standard serum creatinine kidney function tests performed before and after treatment of 32 patients showed no change. Analysis of urine gave normal results in all patients before treatment; after treatment, two patients showed "one plus" of albumin in the urine and two showed a trace of albuminuria. Cephalin flocculation, thyroid turbidity and thyroid flocculation tests carried out in 36 patients and bromsulphophthalein tests made in 15 patients showed no evidence of liver function abnormalities after prolonged administration of griseofulvin. The only side effects noted were four cases of headache, one of abdominal cramps and one of slight insomnia. All these manifestations disappeared whilst the patients were still under therapy with griseofulvin. Tinea capitis, favus, tinea corporis and tinea cruris were found to clear readily. The results were excellent in tinea manum and onychomycosis of the finger nails when griseofulvin was given for a prolonged period. The authors considered it doubtful whether griseofulvin treatment would prove to be satisfactory for the majority of patients with onychomycosis of the toe nails and tinea pedis. They conclude that griseofulvin appears to be an effective, safe and relatively non-toxic antibiotic in the treatment of various types of superficial human fungous infections.

Long-Term Oleandomycin Therapy for Acne.

C. STUTZLER (*A.M.A. Arch. Derm.*, June, 1960) discusses a series of 200 patients with severe acne who were started on long-term therapy with triacetyl oleandomycin; 16% of these completed treatments lasting periods of six months to two years. Of these, 88% showed definite improvement. Only 7% discontinued treatment because of various sequelæ, and most of these were improving at the time treatment was discontinued. Clinical evidence is presented that there is little tendency for the emergence of oleandomycin-resistant organisms in these patients. The author was unable to find any evidence that the prolonged use of triacetyloleandomycin has any influence on the incidence of upper respiratory infections, including acute tonsillitis, acute otitis media in adults, acute gastro-intestinal infections, or furunculosis. The possible role of nasal staphylococci in the maintenance of acne and the possible usefulness of applications of antibiotic ointments and creams to the nasal vestibule as a treatment adjuvant, as a possible means of diminishing flares and recurrence, and even as a possible means of preventing severe acne in adolescents, are considered.

Dermatitis with Capillary Fragility.

R. GREENWOOD (*A.M.A. Arch. Derm.*, June, 1960) discusses dermatitis with capillary fragility and states that cases of Schamberg's type are rarities, and that in those which are seen many causes may be operative. However, it is clear that acute, subacute and chronic dermatoses, associated with increased capillary fragility, have occurred in relation to the wearing of wool clothing. Dermatitis patients with capillary fragility of Schamberg type have been examined with a view to their sensitivity to "oils" used in wood processing. Six or seven patients showed definite positive results. The ætiology of Schamberg's disease and its relationship to wool contact are discussed.

The Treatment of Dermatomycoses with Griseofulvin.

V. PARDO-COSTELLO (*A.M.A. Arch. Derm.*, May, 1960) remarks that griseofulvin is undoubtedly the most important addition to the therapy of superficial mycoses. It is the drug of choice in tinea capitis, onychomycosis, tinea cruris and tinea pedis. Local treatment may be added with advantage and continued for some time in order to prevent relapses and recurrences and reinfections. Most patients tolerate the drug well. No blood changes or liver or kidney disturbances were observed in the author's series of cases. Patients can continue their daily occupations without any discomfort. There were three patients who developed a morbilliform rash after twenty-four hours of medication. One of them was a chronic alcoholic, known to be allergic to penicillin, the others were healthy young men. Relapses and reinfections are common unless griseofulvin is continued for several weeks after apparent clearing of the lesions. The author believes that a powder or an ointment containing one or more of the well-known chemical anti-fungal drugs could be used with advantage in conjunction with griseofulvin, except in cases of tinea of the scalp and onychomycosis, in which they are unnecessary. Moniliasis developing as a complication of tinea cruris and of tinea pedis was observed in a few cases and easily controlled. The most outstanding achievements of griseofulvin are in the treatment of hitherto recalcitrant *Trichophyton rubrum* infections of the skin and nails, and in tinea capitis.

In-Vitro Resistance with Griseofulvin.

R. C. V. ROBINSON *et alii* (*A.M.A. Arch. Derm.*, May, 1960) conclude that *Trichophyton mentagryphytes, T. rubrum, T. tonsurans, T. Schönleini, Epidermophyton floccosum, Microsporum audouini* and *M. canis* apparently developed resistance to griseofulvin *in vitro* when exposed to the drug in increasing concentrations. The existence of resistant stains of dermatophytes *in vitro* suggests that in-vivo resistance may develop. There is no apparent relation of in-vivo response and in-vitro response to the drug.

Tumours of the Palm and Sole Resembling Basal Cell Epithelioma.

G. A. HUNTER AND F. F. HELLIER (*Brit. J. Derm.*, August-September, 1960) describe two tumours, one on the palm and the other on the sole, which resembled basal cell epitheliomas. Histological examination suggested that they were derived from the sweat duct apparatus and possibly from the acrosyringium as suggested by Binkas. These tumours may sometimes have a rather characteristic clinical appearance. The lesions grow very slowly; in one of the authors' cases the tumour had been present for 30 years.

Behcet's Disease as a Multiple Symptom Complex.

C. BERLIN (*Arch. Derm.*, July, 1960) gives a report of ten cases of Behcet's disease, two of which are described in detail. The disease is of multiple manifestations, involving joints, skin (predominantly of the genital region), mouth, eyes and sometimes brain and viscera. Vascular changes probably represent the basis of the morbid process which is allergic, inflammatory and mostly ulcerative in character. The course of the disease varies from a relatively benign one, ending in virtual cure, to a malignant form which may end fatally. The course may be latent or active; the active stage is manifested by acute episodes of the various symptoms which recur singly or simultaneously at irregular, frequent or rare intervals. There is no specific remedy. The disease is characterized by a peculiar reactivity of the skin to an intradermal saline injection and to a needle puncture.

Liquid Nitrogen in the Therapy of Skin Lesions.

A. F. HALL (*Arch. Derm.*, July, 1960) states that the very cold liquefied gases, in his experience liquid nitrogen, provide a superior method of treatment for lesions which consists primarily of an avascular keratotic acanthotic mass resting upon a normal vascular bed. Such lesions can be frozen without pressure so that the underlying normal vascular bed is not frozen, and the line of vesiculation appears between the lesion and the normal underlying tissue. This is followed by little or no scarring, in contrast to the sequelæ of freezing the underlying normal tissue along with the lesion, as occurs when the freezing is accompanied by pressure. The author names the lesions which are appropriate for treatment, and these are as follows: warts, X-ray keratoses, senile and seborrhœic keratoses, and in selected cases, Bowen's disease, hypertrophic scars (recent), granuloma annulare, discoid lupus erythematosus, pigmented nævi and pseudomucinous or synovial cysts at finger joints.

Solitary Trichoepithelioma.

I. ZELIGMAN (*Arch. Derm.*, July, 1960) states that true solitary trichoepitheliomas make their appearance at any time from

birth to the fourth decade. There is no history of familial occurrence. Thus far they have only been noted on the face and are relatively small, usually not over 2 cm. in diameter, and are asymptomatic. The lesions are usually round and papular. At times they are annular. In the differential diagnosis the following have to be considered: granuloma annulare, pigmented and non-pigmented nævi, and basal-celled carcinoma. Microscopically, the important problem is that of separating hæmartomatous trichoepithelioma from basal-cell carcinoma differentiating towards hair follicles. Sometimes this is impossible and clinical data may have to be relied on. It is evident that solitary trichoepitheliomas should be kept in mind when a single solid or annular papule appears first prior to 30 years of age. Confirmation by biopsy is essential.

UROLOGY.

"Madribon" and Pyridium in Urinary Infections.

B. PINCK (*J. Urol.* (*Baltimore*), July, 1960) states that pyridium (phenylazodiamino-pyridine) has been reported to potentiate the in-vitro antibacterial activity of the sulphonamides. However, its main value in urinary tract infections appears to be its analgesic effect on urinary tract mucosa. This action occurs quickly, long before any chemotherapeutic action could control the infection. The sulphonamide used by the author in this study is sulpha-dimethoxine ("Madribon"), which has a wide range of antibacterial action. It is slowly excreted in the urine as the soluble glucuronide, and maintains therapeutic blood levels for long periods on low dosage. Paraplegic patients on this drug continually for up to one year have shown good tolerance. Vitamin B complex is given as an adjunct when indicated. Fifty-four patients with a variety of urogenital disorders were tested. The organisms cultured were of all kinds, including the very resistant *Proteus* and *Pseudomonas*. Mixed infections were present initially in well over one-third of the cases. The therapeutic trial was carried out by a five-day course of capsules combining 125 mg. of "Madribon" with 50 mg. of pyridium. Two capsules were given every six hours. Effectiveness was evaluated on a basis of clinical remission, the results of bacteriological examinations and the finding of effective levels of "Madribon" in the urine. On this basis, the therapy was effective to varying degrees in 90% of patients.

Prognosis of the Solitary Kidney.

J. E. DEES (*J. Urol.* (*Baltimore*), May, 1960) states that the urologist is often asked by a patient on whom he has performed nephrectomy what is his outlook for the future. If the remaining kidney is normal, most urologists assure the patient that his outlook is a normal one. The author has reviewed 166 cases of nephrectomy to determine what happened to the patient afterwards. Of the entire group of 166 patients, 16 already had disease in the opposite organ at the time of operation, and in 29 trouble developed during the follow-up period. The period of observation was three years or more. In another group of 33 patients with congenital solitary kidney some disease developed in 22.

Retropubic Prostatectomy with No-Catheter Technique.

L. S. DEBENHAM AND A. E. WARD (*Brit. J. Urol.*, June, 1960) describe their experiences in retropubic prostatectomy without tube drainage of the bladder either per urethra or suprapubically. By eliminating such a foreign body it is hoped to avoid post-operative infection. The authors consider that the development of an extreme hypotensive technique of anæsthesia has raised the possibility of employing a no-catheter technique from 60% to 80% of cases. The systolic pressure falls to between 50 and 80 mm. of mercury. By the end of the operation, the pressure is slowly beginning to rise. In cases where the post-operative systolic pressure does not commence to rise after three hours, a nor-adrenaline drip infusion is given. A catheter is left in at operation in the following circumstances: (i) with diabetic patients, for whom four-hourly specimens of urine are needed for their post-operative management; (ii) after undue bleeding at operation; (iii) if the capsule is not water-tight after suture; (iv) after unduly prolonged pre-operative suprapubic drainage. Eighty-one patients (comprising 78% of a consecutive series) were sent back to bed without an indwelling catheter, and normal micturition followed in all but seven cases. The bloodless field allowed perfect hæmostasis in most cases. The trigonal bar is cut very deeply and the bladder mucosa sutured down as far as possible to the posterior aspect of the prostatic capsule. The prostatic capsule is sutured with complete watertightness. All sutures are of chromicized catgut. This watertightness, as well as the efficacy of hæmostasis, is tested by saline washouts through a catheter, which is withdrawn after five ounces of saline are injected into the bladder. The patient passes urine after an average time of nine hours (shortest four and a half hours, longest 22 hours). As a rule, a catheter is not passed unless the patient is uncomfortable.

Cycloserine in Recurrent Urinary Infections.

R. D. HERROLD AND N. KARABATSOS (*J. Urol.* (*Baltimore*), July, 1960) state that the need for prolonged administration of an anti-infective agent is obvious in many chronic and recurrent urinary tract infections. Many of these resistant infections must be associated with chronic and progressive pyelonephritis, but this cannot be proved without renal biopsy or autopsy. The authors state that they were so impressed by the use of cycloserine ("Seromycin") in acute infections that they decided to try it for long-term therapy. All patients were completely studied first, and all had had many recurrent episodes of acute exacerbation of their infection. With few exceptions there were no associated congenital anomalies. There were 41 patients with non-tuberculous infections. The patients' ages varied from 4 to 82 years. For adults the initial dose was 250 mg. by mouth every 12 hours. This was continued till pyuria disappeared, usually in 2 to 3 weeks, after which the dose was reduced to 250 mg. once daily. In children the initial dose (100 mg. twice daily, even for children of 4 to 6 years) was continued for 3 to 6 months, and this was well tolerated. Cultures were made frequently during medication. "Positive" cultures sometimes occurred, even though pyuria and symptoms had disappeared; this occurred especially when the pathogens were *Proteus*, *Pseudomonas* or enterococci. However, many of these "positive" cultures were very weak. Some of the advantages of cycloserine are absence of gastro-intestinal side reactions and an almost complete absence of skin reactions. It has been found that neurotoxic reactions even with doses of 2 grammes per day are markedly reduced when pyridoxine is given in a dose of 300 mg. per day. Bacteriostasis in the urine lasts for 24 to 48 hours after the antibiotic is discontinued.

Post-Voiding Films in Excretion Urography.

D. R. ROONEY (*J. Urol.* (*Baltimore*), August, 1960) states that urologists and radiologists are often confronted with the problem of non-visualization of one ureter in excretion urograms. This is usually an effect of obstruction. A technique is advised for opacifying the ureter which does not show up. This is simply to get the patient to empty the bladder after walking to the toilet. He then walks back to the X-ray room, and an ordinary supine film is made. If no opacification of the ureter concerned occurs, then another film is taken after voiding an hour later. This has been helpful, not only in more promptly opacifying the obstructed ureter, but in frequently disclosing the exact site of obstruction by opacifying the ureter right down to the obstructing lesion or body. Several mechanisms probably interact in helping to opacify the obstructed ureter. (i) By getting the patient upright to walk, the effect of gravity is utilized. (ii) If a nephrographic effect (the nephrogram phenomenon, or opacification of the kidney itself) is looked for, it is possible to predict roughly how long it is going to take to opacify the ureter. Thus, if the nephrogram is slow in appearing, the post-voiding effect on the ureter is looked for later. The author has observed an obstructed ureter to take 24 hours before opacification first occurred. (iii) The action of voiding itself seems to exert an effect on the opacifying of the ureter. Patients have been observed in whom only the calyces were opacified in the routine films, or even in delayed films, and yet, in less than five minutes after voiding the ureter would be opacified right down to the obstructing lesion. It is suggested that voiding may reflexly stimulate peristalsis in the upper part of the tract. Another possibility is that movement of opaque urine down the ureter is a passive process made possible by elimination of urine below the partial obstruction. Another useful purpose of the post-voiding film is that it permits visualization of the terminal few centimetres of the ureter, previously obscured by the opacified bladder. This is the region where most calculi lodge.

Brush Up Your Medicine.

METHODS OF TREATING OTORRHŒA AND SOME COMMON DISEASES OF THE EXTERNAL EAR.

THROUGHOUT medical history there has always been a tendency to repeat the statements of the "authorities" without criticism. Probably nothing has retarded medical progress more. The tendency has been particularly marked in therapeutics, and even today, when this should be based on physiological pharmacology, the old traditional remedies are being prescribed, even when it is known that the agent prescribed is inert, ineffective or even harmful. For example, babies are put into mustard baths for convulsions, and gargles are prescribed for sore throats, although we know that no gargle passes beyond the anterior faucial pillars.

It seems reasonable to me that our therapeutics should be dictated by an accurate diagnosis, physiological pharmacology and reasonable action to see that our therapeutic agents are enabled to be effective. Our steps in treating any illness should be: (i) diagnosis—(*a*) ideal, (*b*) practical; (ii) treatment by known effective agents and procedures; (iii) ensuring that the agents can act effectively.

The foregoing remarks particularly apply to otorrhœa and other diseases of the external canal. Before one can make a diagnosis, one must be able to see the whole canal.

In order to examine and treat an ear satisfactorily a toilet may be needed. This can be done with a Jobson Horn probe, dressed probes or syringing. I rarely use a syringe, as water is contraindicated in some ear conditions. With a clear view the condition can be diagnosed and treated reasonably. Putting drugs into an ear containing débris and pus means that some of the infecting organisms are hidden safely and can reproduce themselves the moment the environment becomes favourable. When pus is present, the ideal is to make cultures and then supply a suitable therapeutic agent; but in practice this is rarely necessary, and the patient expects some immediate attention and treatment, if only for his pain. Thus one makes an immediate provisional diagnosis, and this can be confirmed by suitable investigation, such as identification of the infecting organisms and antibiotic sensitivity tests.

My object in presenting this paper is to advocate a method of treating tropical ear and otitis media. In order to consider the subject with proper balance I will briefly refer to other ear complaints, with the object of ensuring a definite diagnosis.

Allergic Dermatitis: Otitis Externa.

When a patient complains of otorrhœa or symptoms located in the pinna and external canal the commonest possibilities are, starting from without inwards, (i) allergic dermatitis or eczema and (ii) otitis externa circumscripta or furunculosis.

Allergic dermatitis or eczema of the pinna, concha or external canal, if of long standing and with exudate, is usually an allergic condition with possibly secondary infection. The treatment of this condition when it affects the pinna or the concha is that of allergic dermatitis anywhere. We shall consider allergic otitis externa in connexion with otitis externa in general. The scalp of the patient or the spouse usually displays dandruff or possibly seborrhœic dermatitis, and it is essential that this be treated also. A nightly application of *Lotio Capillaris* R.P.A.H. or the more expensive "Selsun" (Abbott) is usually sufficient for the scalp, and Ung. "Pragmatar" ointment or ether-soluble tar paste (Martindale) for the pinna. If these prove irritating after a few days, a change is made to *Cremor Zinci*, and in rare cases to cortisone ointment.

The next condition to consider is furunculosis. This must occur only in that part of the meatus where there are hair follicles. With much swelling it may be difficult to distinguish from otitis externa diffusa, but after the meatus has been packed with anhydrous glycerol the swelling and pain subside, and the deeper canal and membrana tympani can be seen free from inflammation. An obvious furuncle may be seen, or there may be one spot, tender on pressure with a probe or speculum, and swelling is found.

Treatment consists of the administration of procaine penicillin, one megaunit per day, with, if the furuncle is discharging, Sulzberger's powder to reduce the risk of a new hair follicle becoming infected. Where the furuncle is pointing or discharging, pus can be obtained for antibiotic sensitivity tests.

Tropical Ear; Otitis Media.

We are now left with two common causes of otorrhœa, otitis externa diffusa or tropical ear and otitis media with perforation. It is to the former of these that I make special reference. It is easily treated, provided an ætiological diagnosis is made. It is very common, and is mainly due to (*a*) bacterial invasion, (*b*) fungous infection, (*c*) allergy, (*d*) allergy complicated by (*a*) and (*b*). Most commonly it is bacterial in origin, and *Pseudomonas pyocyanea* and *Staphylococcus aureus* are the commonest organisms; but the infection is usually mixed, and many other organisms may be present, some of the saprophytes producing a foul smell.

Apart from its cause, otorrhœa may be classified as (i) inaccessible to treatment and (ii) accessible to treatment. If it is inaccessible, the cause will be swelling, and the first step to take is to reduce the swelling. The ear must be packed with narrow selvage gauze, soaked in anhydrous glycerol and replaced often. In 24 hours this will reduce the swelling and abolish the pain. The ear is never so swollen that a pack cannot be inserted. Anhydrous glycerin is not always obtainable, as glycerin is highly hygroscopic and soon becomes diluted in a stock bottle. However, "Sedonan" (Knapp) has, as a vehicle, anhydrous glycerol, and the problem is solved by its use. We have now only to consider cases as accessible. We can perform a toilet, inspect the external canal and perhaps make a provisional ætiological diagnosis. The colour of *Ps. pyocyanea* or *Aspergillus niger* may be seen, or the shape of the fungus components detected. Some strains of *Ps. pyocyanea* are colourless. Pruritus is present in eczema and fungous infections. The intact tympanic membrane can be seen, and this excludes otitis media. If no positive diagnosis can be made, it is best to assume that the commonest cause is operating—*Ps. pyocyanea*.

Patients want action and will not wait for a pathological report. I may say here that I do not use auristillæ. The external auditory canal is lined with modified skin and is meant to be dry. Water as a vehicle in auristillæ, or plain, is anathema to the external canal, and powders give much better results. Indeed, water often irritates otitis externa. *Pseudomonas pyocyanea* requires a high pH for survival, and a low pH is best secured by packing the ear full of lead subacetate powder (1%) in boric acid. If the ear is packed full of powder and kept full, a bulky source of solute is provided with the discharge acting as a solvent, and the mechanical result is better, the application being constant. Auristillæ soon run out of an ear and are washed on to the floor of the external canal, leaving the roof and the walls untreated.

If it is thought that the infection is caused by a fungus, the ear is insufflated with acetyl salicylic powder (1%) in boric acid, or if it is running freely, it is packed with this powder.

If the external otitis is of purely allergic origin, the exudate will be a colourless serous one. Either powder is very effective in preventing or dealing with a secondary infection, whatever it is, and the ear cannot be irritated by the further entry of external allergens if it is completely filled. When applying these powders, I advise the patient to have the new powder packed in over the old and to use no lavage. When pain is severe acetosal compound, with one-quarter of a grain of codeine if necessary, given every three hours, gives relief. This treatment clears the condition in 95% of cases, and the remainder require attack with staphylococcal toxoid, silver nitrate, ionization and finally prayer. One sees at times a dry, scaly eczema of the external canal; it will yield to ether soluble tar paste (Martindale) or "Pragmatar" ointment.

There is a fashion to treat almost any otorrhœa with antibiotics, given either topically or by injection. This urge should be resisted, as there is a danger of sensitization, infection with resistant organisms and disappointment. Penicillin is commonly used, but it has a narrow spectrum and is not effective against the commonest infecting organism, *Ps. pyocyanea*. The antibiotics are not generally successful in chronic conditions.

Otitis Media with Perforation and Otorrhœa.

As we are discussing the running ear, the acute condition prior to rupture will not be discussed, but otorrhœa following rupture of the tympanic membrane will be.

In the acute or recent stage, the first thing is to make a diagnosis. Clean the ear by swabbing, and see the pus or mucus running or pulsating through the perforation. If the perforation cannot be seen, the pulsation is enough, and Valsalva's procedure or the use of Siegel's speculum will often indicate the site.

If the pain has not gone and bulging is still present, or if there is a nipple perforation, myringotomy should be done. Never hesitate. A myringotomy can do no harm.

At this stage it is a good idea to assess the situation. (i) If the discharge is very copious, the area of mucosa in the middle ear is not large enough to account for it, and its source is probably (*a*) the mastoid, or (*b*) the tuba auditiva. The mastoid may be excluded by X-ray examination and the tuba auditiva by examining the naso-pharynx for adenoids or polyps and the paranasal sinuses for chronic sinusitis. The commonest causes of chronic, copious or recurrent otorrhœa from the middle ear are mastoid infections, naso-pharyngeal infections or chronic sinusitis, and these must be treated to achieve success. We must seek the cause and remove it, and the result will be eliminated. (ii) When the middle ear alone is the source of the otorrhœa, the discharge is not copious—indeed, it is often scanty. It may be kept going by a carious ossicle, granulation tissue, a polyp, cholesteatoma, tuberculosis, etc., and these will require the appropriate treatment. In most cases it can be treated and cured simply, and with granulations and polyps removed, persistent treatment will produce surprising results. R. R. Wood of Dublin states that one should achieve dry ears in over 80% of cases.

The most effective treatment is Sulzberger's powder. This is boric acid with 0·75% to 2% iodine worked into it. Sulzberger's powder is not boric iodide powder, and the latter cannot be substituted for it. It is impossible to mix crystalline iodine and boric acid to make an even mixture, and the iodine crystals, however small, will blister the external canal. Sulzberger's powder is made by dissolving the required amount of iodine in ether, grinding it with boric acid in a mortar then allowing the ether to evaporate. Sulzberger's powder must always be made *secundem artem*, and must never be brown in colour. It is used to pack the external canal full once or twice a day after an initial ear toilet. After a week's treatment the ear is generally so much improved that one can start treating the polyp or granulation tissue, if present, with a snare or fused silver nitrate. I would recommend this latter only to those who are used to manipulations in the ear, as one cannot apply it except expertly and under clear vision. If it is applied freely or too high, it can affect the seventh nerve in the Fallopian canal and even produce paresis.

Sulzberger's powder should be persevered with for six weeks if necessary, and ideally should be applied daily by the doctor himself or by a specially trained nurse. If it is not applied effectively, it produces no results; but there is no harm in its use by unskilled hands, when it often succeeds. The untrained can rarely treat infants successfully, so these should be admitted to hospital.

I have tried every ear powder and drop I have ever heard of, but I have never found anything so successful as Sulzberger's powder in chronic otitis media with perforation. However, it is quite unsuitable for otitis diffusa, as it is irritating in these conditions.

When after extended treatment there is a discharge of clear mucus from the middle ear which cannot be abolished, the discharge is a hypersecretion of mucus from the tuba auditiva, and it is probably the best result obtainable. The ear will be reinfected if left alone; but a daily or even weekly insufflation of Sulzberger's powder through a drinking straw or a vulcanite insufflator will prevent this.

If after all trials purulent otorrhœa is still present and the naso-pharyngeal and sinus infections have been cleared up, the next consideration is a mastoidectomy, cortical, radical or modified radical. Every endeavour should be made to achieve a dry ear, as a patient with purulent otitis may always be insidiously approaching vestibular or intracranial complications.

I should like to mention the running ear in children and infants. It is nearly always connected with infected adenoids and/or sinuses. It should always be cleared up. Much adult deafness, including chronic progressive deafness, originates with the otorrhœa of childhood. As a preliminary, adenoidectomy and proof puncture should be done. To succeed in applying local treatment to an infant's otorrhœa, the child should be bundled and the head held by one person while the other does the toilet and applies the powder, always completely filling the ear.

Conclusion.

I do not claim that this short paper is comprehensive; but it does deal with the commoner ear discharges and suggests a systematic approach to their diagnosis and treatment.

The commonest errors in treating the running ear are as follows:

1. Failure to treat it on an ætiological basis.

2. Failure to carry out an effective ear toilet.

3. Failure to treat it on a basis of physiological pharmacology.

4. The use of auristillæ with water as a vehicle.

5. The use of pigments, such as gentian violet. These are not antiseptic in ears, soil the pillow and obscure the view. To some this may seem their main attraction.

6. The use of glycerin of ichthyol. Ichthyol is pharmacologically inert, and its unpleasant colour offends the patients. It is in the same category as vipers' tongues, newts' livers and the caul.

7. Dependence on the local and parenteral use of antibiotics. (*a*) The use of penicillin and sulphonamide powders in the external canal. They are ineffective against many organisms, particularly the commonest found in aural infection, *Ps. pyocyanea*, and no surer way of sensitizing patients to both drugs could be devised. (*b*) Parenteral penicillin injections, which are rarely effective and can also produce sensitivity. The diagnostically destitute and the lazy or overworked immediately use penicillin, forgetting its narrow spectrum and the patient's possible existing sensitivity. The attitude of mind should be one of opposition to the use of antibiotics.

8. The misuse of an aural syringe. Lavage will reinfect a middle ear through a perforated membrana tympani and irritate allergic otitis externa.

R. PALMERSTON RUNDLE,
Former Ear, Nose and Throat Surgeon,
Rockhampton Base Hospital,
Rockhampton, Queensland.

Out of the Past.

COLONIAL BEER.

[From "Colonial Beer", by Dr. James Jamieson, *Melbourne Review*, July, 1878.]

THE amount of beer consumed in Victoria must be looked on as very large, when the circumstances are taken into consideration. A very close approximation may be obtained by adding to the produce of Victorian breweries the proportion of imports retained for home consumption, and deducting from this total the quantity of native production exported, which latter item might almost be disregarded on account of its insignificance. Taking the average of the last five years, the consumption of beer in this colony annually is rather more than fourteen million gallons (14,051,420), and calculating the population of these years as averaging about eight hundred thousand (800,000), we get an allowance of about seventeen and a half gallons per head. This is a very large quantity, both absolutely and relatively to the amount consumed in the other countries mentioned, when we take in to consideration these circumstances:—First, that nearly all the beer consumed here is very strong; second, that all the better sorts, and especially the imported, are very dear—out of all proportion to their intrinsic value; and, third, that a great deal of it is of very poor quality. . . .

[The author proceeds to examine the methods of producing beer and the "diseases of beer" in the light of M. Pasteur's recent work on the subject. After mentioning that beer, like most things, must be mixed with brains, he concludes as follows.]

Anyone, therefore, who reads this book of Pasteur's intelligently, may be able to do a great deal to guard against the introduction of morbific matters, even without making a complete change in his appliances and modes of manipulation, and thus produce an article better to begin with, and more durable, to the benefit both of himself and his customers.

It would be possible to make beer which, being of slight alcoholic strength, could be made of malt entirely, at no great cost, and which could also be allowed to mature properly without great risk of loss from deterioration. Such a beverage, scarcely at all an intoxicant, whilst midly stimulating and tonic, would certainly be much better adapted for general use in Australia than the imported beer, which must be strong to insure its keeping, and most of that manufactured here, which is generally a not very successful imitation of it.

Correspondence.

AN APPEAL.

SIR: I would be grateful if through your columns you could be of assistance to our nun doctor, Sister M. Leo, M.D., who for the past nine years has carried out medical work in New Guinea. Samples of drugs, creams, lotions, soaps or anything medicinal would be useful to her in her work amongst the natives of New Guinea. Sister M. Leo is a graduate in medicine of Marquette University, Illinois, U.S.A.

If anyone reading this appeal would have any drugs or instruments for which they no longer have any use and would like to donate them to Sister M. Leo, they would be gratefully accepted at this hospital, and we will forward them on to her.

Yours, etc.,

SISTER M. OLIVER,
Matron.

Missionary Sisters of the
Society of Mary,
"Lourdes" Hospital,
95 Stanhope Road,
Killara, N.S.W.
December 14, 1960.

DEVELOPMENTS IN OUTBACK MEDICAL SERVICES.

SIR: Following your excellent editorial "Developments in Outback Medical Services", could I answer Dr. C. R. R. Huxtable and try to fill in some of the details of his excellent, provocative, but rather bald letter of November 19, 1960? It raises so many issues.

Even after six years away from the Broken Hill Base, I still get excited and angry. As it stands, the Royal Flying Doctor Service is a magnificent service, and has achieved so much; such hackneyed phrases as a practice covering 250,000 square miles become quite alive when you are left to your own devices and have to look after them. Still it is too true that a service cannot be run by wise and noble idealists 700 miles from the scene of action without many unfortunate happenings. Liaison is bad. The field always has felt it is not understood by headquarters. (Can I recommend Dr. Huxtable to read the correspondence from another very famous Flying Doctor, Dr. Woods?) Broken Hill was a continued sequence of petty problems that our remoteness converted to gross injustices. But at least the executive was not all the time interfering or harping or skimping. And besides, we grew. Out of 1000 outpost stations throughout Australia linked to eight bases in 1954, we in Broken Hill had 250 attached to us—a fact which Dr. Huxtable must remember when he complains about expenses. We were flying two days out of three.

About night flights. Dr. Huxtable quotes 1954 as the shibboleth. There were two night flights in the first half of 1954, since when apparently there have been no more by aircraft of the Royal Flying Doctor Service—wisely, I think.

Night flying is inherently more dangerous and demands adequate technology. It does not at present exist in these areas. The only aerodrome that could cope is Broken Hill. This is the real reason for only one base in New South Wales. Bourke is not adequate.

In 18 months at Broken Hill there were only four real occasions for night flying. Once I was at Tibooburra and had a very sick child at Canopus in South Australia, near the Murray, with croup. We did not fly. The child settled at home with adequate steam only after many anxious hours. Again, the manager's wife at Glengyle Station, 100 miles north of Birdsville, called me late at night about a woman 30 miles away who I felt had a leaking ectopic. We flew at 4 a.m. next morning, and after 12 hours' travelling had her back at Broken Hill Hospital. They removed a carneous mole from the left tube one Aschheim-Zondek and one month later.

The other two occasions were the reasons for the two night flights: one to a man who had a bullet in his left kidney acquired from a drunkards' brawl on election day, and the other to a man who had his pelvis crushed by the tractor he was driving. He suffered a massive retroperitoneal hæmorrhage. Certainly neither would have stood 200 miles of road transport; and would anyone like the thought of jogging an ectopic 500 miles by road?

Nothing short of a technological miracle would have tempted me to fly to Glengyle at night, and after the night flight to the crush injury, both the pilot and I were cured of night flying with things as they were. It was too hazardous.

Firstly, airstrips. In any case we did not fly to Tibooburra at night, as their airstrip has very dangerous approaches, and the commercial airlines had discontinued regular services there because of this inadequacy. We flew instead to Mt. Brown, a good strip well maintained by its owner. He lent his utility, and so it was overland 30 miles to Tibooburra. The man was transfused and transported by air next morning from Tibooburra to Broken Hill.

Secondly, aircraft. The final flight towards Wilcannia was delayed one hour, as there was a radio blackout on 72 metres, our most used wave-length. We landed on a fair but untried airstrip to the glare of home-made rag and petroil flarepots. When we took off with our patient to return to the Hill, the moon had dropped, and the wind had changed. Another flarepath had to be set out, the aircraft nearly bogging taxiing over to it. The takeoff was most unusual, with the plane circling one wing low for a while. The "turn and bank" indicator had gone "on the blink" on takeoff, and with the artificial horizon unserviceable from the start of the flight, the pilot had to take off with an invisible natural horizon and no artificial aids to altitude. Still, by superb flying we reached the Hill without further incident.

The Department of Civil Aviation was very helpful, but their regulations do stipulate a dual control aircraft and certain basic navigational aids for regular night flying, thus excluding the Drover. Suitable aircraft are non-existent. The Drover was tailor-made by De Havilland, and he sold some two score, but when early on the propellers shed odd blades De Havilland raised their hands in pious horror, shoved on fixed-pitch propellers and forgot about the unfortunate aircraft, now a very sorry jade. It took seven yeas' hard work to get them to allow the service to use other engines and reliable, well-tested, variable-pitch propellers. It is again a first-rate aircraft, but for daylight flying only.

Thirdly, navigation. Both these flights were done by dead reckoning, compass and clock, no homing beams as used by interstate jets; just that, and the hope that one of the homesteads will hear you overhead and report your position to you. In the daylight it is easier. Every 30 or so miles or more you see a roof that is recognized or a sandhill that is the right colour: yellow at Innamincka and rich red at Arrabury. Even the change in a roof colour can throw you out if you do not know of the change. The vastness and sparseness cannot be over-estimated. The smaller blocks are 20 square miles, whilst Innamincka is a mere 7500 square miles in area with some 21 men employed.

However, the owner of Mount Brown was quite prepared to install and maintain a radio beacon to aid navigation. The solution is expensive, but possible. To run a fully certified night flying aircraft would make the £20,000 running cost, so upsetting to Dr. Huxtable, mere chicken-feed. And as for further bases, he would have a stroke. Doctors, on the other hand, are cheap.

I disagree that Queensland appears to lead in the matter of clinics. Even in 1954 we flew two days out of three, and eight to ten days a month of these were to clinics. There were clinics at Tibooburra, Marree, Andamooka, Finnis Springs, Radium Hill, Menindee, Hungerford and Moombidary. And if they had no doctor, we filled in at Wilcannia and Ivanhoe.

Certainly there is room for expansion. Canopus in South Australia was being tested to initiate a routine call there. The greatest need of all was east of the Darling, which was more isolated than the western side. So many of the small towns can only support one-third or one-half of a doctor. He, therefore, leaves them after a short time and moves on, leaving them once more on their own. So often Ivanhoe, with its small but useful hospital, was served by the doctor from Wilcannia driving over—if there was one there at the time. How much better if these smaller towns could combine and the Royal Flying Doctor Service tend their needs with a second or third doctor and appropriate light aircraft. This to me is just as big a future as a flying surgeon or obstetrician.

Certainly this presupposes better liaison with that distant, hard-working group of idealists the New South Wales executive, which has laboured so hard to make this the magnificent service it is. No doubt a second or third doctor would be an enormous help in this regard, helping to keep

the executive in touch. It needs vigorous young men with very old, wise heads—very rare.

It would seem that even young men, not necessarily wise, who are rash enough to leave our capital cities are rare in this age. I think rather it is lack of publicity at the undergraduate level that explains why we needed a migrant to go to Antarctica with an Australian expedition, or for that matter, how a youngster with only one year's country hospital residence was the best offering himself to look after that wonderful quarter of a million square miles.

My apologies for the length of this letter, but your correspondent had raised so many matters, all of which touched the heart.

Yours, etc.,

C. RUSSELL-JONES.

35 Montagu Street,
Goulburn,
N.S.W.
December 14, 1960.

RESTORATION OF DRUG AUTHORITY.

SIR: I desire to inform you that, in accordance with the provisions of Regulation 25 (2) under the *Police Offences (Amendment) Act*, the Minister has directed that the withdrawal of the authority of Dr. J. F. Marrington, 49 Barker Road, Strathfield, to procure and be in possession of drugs to which the Act applies for the purpose of his profession and to issue prescriptions for such drugs shall cease to operate.

The restoration of the authority is effective as from December 19, 1960.

Would you please be good enough to notify the restoration of this authority in your journal?

Yours, etc.,

A. G. KINGSMILL,
Under Secretary.

Chief Secretary's Department,
121 Macquarie Street,
Sydney.
December 12, 1960.

A POINT OF ETHICS: EXAMINATION FOR INSURANCE COMPANIES.

SIR: I would like to request through your columns that medical officers examining patients (especially my patients) at the request of insurance companies should carefully distinguish between the cases where their opinion is requested by the doctor actively treating the patient and those in which they are requested to report by and for the companies; also, that they will endeavour to distinguish those cases where the patient is correctly exercising his ethical right to seek further opinion from those where he merely seizes an opportunity adventitiously provided by the insurance company sending him in order to obtain a report for themselves.

Surely it is grossly unethical to offer quite unsolicited advice to the patient of another doctor, especially when such advice may be frankly antagonistic to that already given by the doctor of his own free choice (an opinion confidentially to the insurance being quite a different matter). Certainly it is very bad manners, not to mention meddlesome interference.

Yours, etc.,

C. C. MCKELLAR.

143 Macquarie Street,
Sydney,
December 14, 1960.

SYDENHAM'S CHOREA: ITS RELATIONSHIP TO RHEUMATIC INFECTION AND PSYCHOLOGICAL ILLNESS.

SIR: The article concerning Sydenham's chorea (MED. J. AUST., November 12, 1960) was of great interest, but marred by the absence of one or two simple facts.

At one point it is stated that the average intelligence of the 19 children as assessed by the W.I.S.C. was 91, "which is *significantly* below the *general population* average" (my italics). What is meant by "significantly"? Does this mean statistically significant? If so, what is meant by general population—especially when the tested children had an age range of about four to thirteen years? If the statement made by the authors is to be interpreted statistically, then the mean, the range and the standard deviation should surely have been given. If, on the other hand, the word significantly is not used in a statistical sense, what does it mean—simply that the observed difference between the means was considered a finding of interest?

Again, one reads: "The absence of bright children was statistically significant." Surely not only more complete data should be given as well as the level of significance, but the problem of adequate sampling needs to be considered, if only very briefly.

It seems a great pity that an interesting article should be so easily subject to criticism when a very few extra facts and figures would provide complete immunity. Quite apart from this, it would allow the article to be of greater value to later research workers, who may well be concerned with statistical problems but be quite unable to refer to earlier research that does not give clear evidence—either original data or ultimate deductions.

Yours, etc.,

ALLEN A. BARTHOLOMEW.

4 Adamson Street,
Heidelberg,
Victoria.
December 12, 1960.

A GUIDE TO BLOOD TRANSFUSION.

SIR: The New South Wales Division of the Australian Red Cross Society has published the second edition of the small book entitled "A Guide to Blood Transfusion", by R. J. Walsh and H. K. Ward. The book is intended by the authors to meet the requirements of Australian medical students, general practitioners and others who administer blood transfusions, but who do not need the detail provided by larger reference books on the subject. Some of the weaknesses of the first edition have been eliminated in the new, and additional information has been included.

We are making this book available at its cost to us, and the authors will receive neither royalties nor fees. It is obtainable from the New South Wales Red Cross Blood Transfusion Service, 1 York Street, Sydney, at £1. To avoid opening accounts, it is requested that intending purchasers forward their remittance when ordering.

Yours, etc.,

A. M. MCINTOSH,
Chairman,
Blood Transfusion Committee.

1 York Street,
Sydney.
December 5, 1960.

POLIOMYELITIS VACCINATION.

SIR: I wish to heartily endorse the remarks made by J. F. Gendle in your issue of December 3, *re* the absurd rules and regulations governing the procurement of Salk vaccine.

One of the big points made by the Government is that adequate records must be kept of all who receive the vaccine. Surely, if this is from the statistical point of view, there have now been enough injections given to provide the answers to all possible questions. Therefore, is there now any valid reason why this vaccine (which for so long has held the limelight) cannot be handled in exactly the same way as triple antigen, tetanus toxoid, anti-tetanus serum, or any of the other commonly used vaccines? Let us prescribe them when wanted. This futile direction by the Government is indeed the last straw, and to my mind should be resisted in its entirety. If we agree to this type of direction, then we have no hope of resisting further regimentation.

Yours, etc.,

H. J. MELVILLE.

22 Beveridge Street,
Swan Hill,
Western Australia.
December 12, 1960.

Medical Matters in Parliament.

HOUSE OF REPRESENTATIVES.

THE following extracts from *Hansard* relate to the proceedings of the House of Representatives.

November 30, 1960.

Restrictive Trade Practices.

MR. WHITTORN: My question is directed to the Minister for Health. Are major changes taking place in America with respect to the distribution of drugs and pharmaceutical products, with a view to reducing costs to consumers? If so, are similar changes being effected in Australia? If not, will trade practices in this industry be included amongst those being studied by the Attorney-General in his investigation of restrictive trade practices?

DR. DONALD CAMERON: I understand that in America a Committee of the Senate has been appointed to inquire into the price of drugs. I am not sure whether it will inquire into distribution but certainly it will inquire into prices. I also understand that so far the committee has not made any report. Conditions in Australia are different from those in the United States of America in this respect, and as the honourable gentleman will realize the Commonwealth Government has no direct control over prices. The Government has no direct control of the prices of drugs except by influencing the prices at which drugs can be admitted to the list of pharmaceutical benefits. The Department of Health is active in this regard and, in fact, I think it is no exaggeration to say that many of its actions in the past have saved the taxpayer millions of pounds by keeping the prices of drugs within a reasonable range when they are admitted to the pharmaceutical benefits list.

SENATE.

THE following extracts from *Hansard* relate to the proceedings of the Senate.

November 29, 1960.

Pharmaceutical Benefits.

SENATOR ANDERSON: I preface my question, which I direct to the Minister representing the Minister for Health, by saying that about five days ago the Minister for Health stated that the drug androstanolone would be taken off the free list on 1st January because it was being prescribed for the treatment of minor ailments, whereas it was intended for special cancer treatment. Will the Minister ask his colleague to consider allowing this drug to remain on the free list, with the qualification that it be prescribed only for special cancer treatment? It seems to me that to take it off the list because it is being used to treat people who do not need it will deny it to the people who need it most.

SENATOR HENTY: As I read the statement of the Minister for Health, the drug is not to be taken off the list but is to be made subject to the restriction that it be prescribed only for the purpose for which it is intended—namely the treatment of cancer of the breast. I understand that each application of the drug costs £12 9s. 6d. The Minister has, very rightly, taken steps to ensure that it will not be prescribed for minor ailments when other and far less costly drugs would fill the bill.

Mental Health.

SENATOR HANNAFORD asked the Minister representing the Minister for Health, upon notice:

1. What is the present state of the £10,000,000 fund set up by the Commonwealth Government in 1955, on the recommendation of the Stoller report, to be made available to the States on a £1 to £2 basis for the provision of capital works associated with the improvement of mental health?

2. What percentage of the drawings against this fund has been used to provide facilities for the treatment of mentally retarded children?

SENATOR HENTY: The Minister for Health has now furnished the following reply:

1. Amounts totalling £5,631,313 have been paid to the States under the *State Grants (Mental Institutions) Act*. The Act authorizes payment of further amounts totalling £4,368,687.

2. An appreciable proportion of the grants has been used for the provision of facilities in mental hospitals where mentally retarded children are patients. In addition 1 per cent. of the grant has been applied specifically for the provision of faculties in special treatment centres conducted by the States for the treatment of mentally retarded children.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

Gynæcology and Obstetrics Refresher Course.

THE Melbourne Medical Post-Graduate Committee announces that a gynæcology and obstetrics refresher course for recent graduates will be held from January 31 to February 10, 1961, at the Royal Women's Hospital. The commencement of the course depends on a satisfactory enrolment. Full residence will be available at the hospital; board and lodging at £7 10s. a week is payable to the hospital. If they so desire, candidates may enter the hospital on Monday, January 30. It will be desirable for those taking part to wear long white coats while in the wards. The programme is as follows:

Tuesday, January 31: Introduction, Dr. J. Laver and the Dean; "The Premature Infant", Dr. G. White; "The Third Stage", Dr. N. C. DeGaris; "Gynæcological Bleeding", Dr. C. K. Churches.

Wednesday, February 1: "Analgesia and Anæsthesia", Dr. K. McCaul; "When and How to Apply Forceps", Dr. R. M. Rome; "Ectopic Pregnancy", lecturer to be arranged; "Endocrines in Gynæcology", Dr. J. W. Johnstone.

Thursday, February 2: "Obstetrical and Neonatal Pathology"; Dr. H. Bettinger; "How to Assess Progress in Labour", Professorial Unit; "Diagnosis and Treatment of Conditions of Cervix", Dr. A. M. Hill.

Friday, February 3: "Analgesia and Anæsthesia", Dr. K. McCaul; "Abortions", Dr. W. Rawlings; "Abnormal Presentations", Professorial Unit.

Monday, February 6: "Ante-Natal Care", Dr. M. Mackie; "Preeclampsia", Dr. F. Forster, "Discharges", Dr. A. Long.

Tuesday, February 7: "Infection in the Newborn", Dr. T. Maddison; "Anæmia in Pregnancy", Dr. P. Jeffery; "Pruritus", Dr. H. Hattam.

Wednesday, February 8: "Varicose Veins", Dr. J. Smibert; "Cytology", Dr. B. Anderson.

Thursday, February 9: Quiz session, Professorial Unit; "Rh Immunization", Professorial Unit; "Endometriosis", Dr. V. Hollyock; "Retrospect", the Dean.

Friday, February 10: "Infant Feeding", Dr. K. Campbell; "Pregnancy after Infertility", Dr. G. Ley; "Pelvic Infections", Professorial Unit.

The fee for this course is £10 10s., and should be sent by Monday, January 9, 1961, with the enrolment form, to the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne. Telephone: FB 2547.

Notes and News.

Post-Graduate Training Fellowships in Psychiatry.

The Senate of the University of Sydney has awarded Post-Graduate Training Fellowships in Psychiatry to Dr. A. G. Bennett, of Denistone, and Dr. Eva Mandel, of Woollahra.

Australian Association of Physical Medicine and Rehabilitation.

The fifteenth annual meeting of the Australian Association of Physical Medicine and Rehabilitation was held in Canberra on October 27, 1960. The following office-bearers were elected: President, Dr. A. Burnell (South Australia); Senior Vice-President and President-Elect, Dr. Gordon Rich (Tasmania); Vice-President, Dr. A. Paul (New South

Wales); Honorary Secretary-Treasurer, Dr. L. Wedlick (Victoria); fifth Committee Member, Dr. Naomi Wing (New South Wales).

After the meeting a scientific session was held and the following papers were presented: "Managament of Lesions of the Cervical Spine", Dr. R. F. May; "Ultrasonics", Dr. G. Rich; "Development of a Rehabilitation Centre", Dr. L. Wedlick; "Rehabilitation Techniques", Dr. N. Wing; "International Aspects of Rehabilitation", Dr. S. Nelson.

The Orthoptic Council of New South Wales.

The Orthoptic Council of New South Wales announces that a course of instruction in orthoptics, of two years' duration, will commence on February 6, 1961. Applicants must be aged 18 years, or 17 years with their Leaving Certificate. They should apply to the Secretary, Dr. M. Sterling-Levis, 235 Macquarie Street, Sydney, by January 24, 1961, submitting copies of three original references.

The Drug and Proprietary Medicines Association of New South Wales.

The Drug and Proprietary Medicines Association of New South Wales has elected the following Executive Committee for 1961: President, Mr. W. R. Kerr (Sterling Pharmaceuticals Pty. Ltd.); Members, Messrs. L. Affleck (Beckers Pty. Ltd.), R. E. Gostelow (The Wholesale Drug Co. Ltd.), N. V. Kerr (D.H.A. (N.S.W.) Pty. Ltd.), K. S. Maddox (Watts Winter Pty. Ltd.), J. L. Phillips (Nicholas Pty. Ltd.), and D. A. Toppin (R. D. Toppin & Sons Pty. Ltd.). The Secretary is Mr. F. A. J. Reynolds, Chamber of Manufactures of N.S.W., 12 O'Connell Street, Sydney.

Prize for a Medico-Surgical Film.

The annual prize awarded by *La Presse médicale* for a medico-surgical film, amounting to 1000 NF cash (with the possibility of its being divided), and various other awards, will be presented during the last session of the course of *Actualités médico-chirurgicales* at the *Nouvelle Faculté de Médecine de Paris* on March 14, 1961. The judges will consider the instructional value of the film as well as its properly cinegraphic quality. Only film of 16 mm. size will be accepted. The time of projection must not exceed 30 minutes. Entries and films should be sent before February 1, 1961, to the *Secrétariat du Journal La Presse Médicale*, 120 Boulevard Saint-Germain, Paris VI°. Awards will be made to the authors of the best films. All films may be entered, including those subsidized or produced by a laboratory or a firm.

Commonwealth Director of Tuberculosis.

Dr. Alan King will take up his appointment as Commonwealth Director of Tuberculosis towards the end of January, 1961, in succession to the retiring Director, Dr. Hilary Roche. Dr. King has been Director of Tuberculosis Control in the Western Australian State Public Health Service since 1950.

Increase in Venereal Disease.

A committee of Commonwealth and State Government medical officers has reported to the National Health and Medical Research Council that there has been a sharp increase in venereal disease in Australia since 1954. This has applied in every State except Western Australia, where notifications have tended downward since 1956. The committee was set up by the N.H.M.R.C. last May, to examine reports that venereal disease was increasing.

Details of the committee's findings have been released by the Minister for Health, Dr. D. A. Cameron. Dr. Cameron said that the members of the Council had arranged to study the committee's recommendations in detail, and would discuss them at the next meeting of the Council. Meanwhile the views of authoritative professional bodies would be sought, to determine the most effective means to meet the problem. The Public Health Committee of the N.H.M.R.C. would consider the question at a meeting to be held at Perth in January.

The medical officers' committee reported that after the war there was a steep decline in the incidence of syphilis in countries using penicillin therapy. This apparently engendered complacency. According to the World Health Organization, syphilis had been increasing in the United States since 1954. In 1959, total notifications were 15%

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 3, 1960.[a]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1	1(1)	..	..	..	..	..	3
Amœbiasis	..	..	..	..	2(2)	..	..	..	2
Ancylostomiasis	4	..	..	..	..	..	9	..	13
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	5	9(9)	5(4)	..	..	..	4	..	23
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	..	..	..	..	1	..	1
Encephalitis	..	..	..	2	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	229(115)	76(48)	19(2)	38(15)	20(10)	2	1	4	389
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1	..	2(2)	..	..	..	3
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	1(1)	..	..	..	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	2(1)	..	1(1)	1	3(1)	..	..	7
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	14(7)	..	3(1)	7(5)	..	..	..	24
Salmonella Infection	..	..	..	5(5)	..	..	..	..	5
Scarlet Fever	6(3)	12(7)	..	3(2)	1(1)	..	..	..	22
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	12	..	..	..	12
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	24(13)	16(14)	7(3)	5(5)	8(3)	5(3)	..	..	65
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[a] Figures in parentheses are those for the metropolitan area.

higher than those of the previous year, and the disease now ranked fourth in prevalence amongst diseases notifiable in the United States. In the United Kingdom, by contrast, the number of cases had continued to decline each year from the peak of 17,675 cases in 1946, and the 704 cases reported in 1958 was lower than at any time on record. There had been a steady recent increase in Australia. Notifications in 1950 numbered 1306. They had fallen to 459 by 1954. By 1959 they had risen progressively to 664. The incidence rate per 100,000 of population was 17·2 in 1950, 5·5 in 1954 and 7·2 in 1959. Experience in the States had for the most part conformed to this pattern, except in Western Australia, where notifications had tended downward since 1956. Notifications of syphilis in males outnumbered those in females by four to one in all States in which syphilis is notifiable. In females the highest incidence was in the age group 15 to 24 years and in males in the age group 25 to 29 years.

The medical officers' committee also reported that notifications of gonorrhœa in Australia totalled 3441 in 1950, 1944 in 1954 and 4128 in 1959. The incidence per 100,000 was 45·5 in 1950, 23·6 in 1954 and 44·7 in 1959. That pattern was uniform in the States, except in Western Australia. In the age group 15 to 24 years, notifications rose by 20% in males and 400% in females. By contrast, there was no increase in incidence in males aged 25 to 29 years, and the number of females in that age group fell by 50%. It was generally agreed that notifications of gonorrhœa were not complete. Many general practitioners were unaware that venereal disease was notifiable. Other doctors regarded notification as a breach of faith. Notification was anonymous, the committee pointed out, and that attitude was not warranted.

Honours.

NEW YEAR HONOURS.

THE following Australian medical practitioners have been included by Her Majesty the Queen in the New Year Honours List:

Dr. Alfred Edward Rowden White and Professor Brian Wellingham Windeyer have been created Knights Bachelor.

Professor Sydney Sunderland has been created a Commander of the Most Distinguished Order of St. Michael and St. George.

Dr. Edward Holbrook Derrick has been created a Commander, Dr. Una Beatrice Porter an Officer and Dr. David Ernest Trumpy a Member of the Most Excellent Order of the British Empire.

University Intelligence.

UNIVERSITY OF SYDNEY.

Dr. J. B. Hickie has been appointed to a senior lectureship in medicine in the University of Sydney.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Turner, Trevor Baxter, M.B., B.S., 1959 (Univ. Sydney), 15 Chapel Road, Vaucluse.

Deaths.

THE following deaths have been announced:

DUNCOMBE.—Cedric Duncombe, on December 15, 1960, at Hobart.

BOSSENCE.—William Adamson Bossence, on December 24, 1960, at Kyabram, Victoria.

HAYDEN.—John Gerald Hayden, on December 26, 1960, at Melbourne.

Diary for the Month.

JANUARY 10.—New South Wales Branch, B.M.A.: Quarterly Meeting of Council.
JANUARY 13.—Queensland Branch, B.M.A.: Council Meeting.
JANUARY 16.—Victorian Branch, B.M.A.: Finance Sub-Committee.
JANUARY 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JANUARY 19.—Victorian Branch, B.M.A.: Executive Meeting of Branch Council.
JANUARY 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JANUARY 25.—Victorian Branch, B.M.A.: Branch Council Meeting.
JANUARY 27.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

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ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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